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THE TRANSACTIONS

OF THE

MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH

VOL. XIX.—NEW SERIES

SESSION 1899-1900

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PREFACE

THE present Volume is the *Nineteenth* of the *New Series*, and contains a record of the work done during the past Session.

That work, as hitherto, embraces the communication of Original Papers; the exhibition of Patients, illustrating rare and interesting forms of disease; and the exhibition of Pathological and other Specimens, so essential to the proper understanding of the morbid changes which take place in the human body.

During the past Session Extra Meetings were held for the exhibition of Patients, Pathological Specimens, Instruments, etc. It is hoped that such Meetings will materially increase the usefulness of the Society.

It is believed that the publication of the Transactions in this permanent form will prove a valuable contribution to medical literature, will encourage the Members to take a more active part in the work of the Society, and will tend in no small degree to increase the influence and usefulness of the Medico-Chirurgical Society of Edinburgh.

WILLIAM CRAIG,
Editor.

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	Dr Alex. James, 10 Melville Crescent,	1877
	Dr Allan Jamieson, 35 Charlotte Square,	1876

		Date of Admission.
	Dr Hugh Jamieson, 13 Rutland Street,	1889
105	Dr James Jamieson, 43 George Square,	1877
	Dr R. J. Johnston, 1 Buccleuch Place,	1897
	Dr G. M. Johnston, 7 Wellington Place, Leith,	1888
	Dr R. M'Kenzie Johnston, 2 Drumsheugh Gardens,	1883
	Dr C. Kennedy, 5 Salisbury Road,	1886
110	Dr C. B. Ker, City Fever Hospital,	1894
	Dr George Kerr, 6 St Colme Street,	1885
	Dr J. H. A. Laing, 11 Melville Street,	1889
	Dr H. G. Langwell, 5 Hermitage Place, Leith,	1899
	Dr H. A. Leebody, Corstorphine,	1899
115	Dr Linton, 60 George Square,	1863
	Dr Harvey Littlejohn, 1 Atholl Crescent, *Treasurer*,	1889
	Professor Sir Henry D. Littlejohn, 24 Royal Circus,	1853
	Dr R. A. Lundie, 55A Grange Road,	1883
	Dr P. M'Bride, 16 Chester Street,	1879
120	Dr W. A. M'Cutchan, County and City Asylum, Burghill, Hereford,	1897
	Dr William C. M'Ewan, Prestonpans,	1892
	John M'Gibbon, Esq., 55 Queen Street,	1868
	Dr John M'Gibbon, Junr., 55 Queen Street,	1899
	Dr MacGillivray, 15 Charlotte Square,	1877
125	Dr G. Mackay, 74 Bruntsfield Place,	1878
	Dr George Mackay, 20 Drumsheugh Gardens,	1887
	Dr Robert Mackenzie, Napier Villa, Merchiston,	1893
	Dr W. Leslie Mackenzie, 4 Summerside Place, Leith,	1895
	Dr William A. Mackintosh, 3 Park Terrace, Stirling,	1896
130	Dr J. Shaw M'Laren, 14 Walker Street,	1887
	Dr P. Hume Maclaren, 1 Drumsheugh Gardens,	1868
	Dr John Macmillan, 48 George Square,	1897
	Dr John Macpherson, 8 Darnaway Street,	1891
	Dr John MacRae, Lynwood, Murrayfield,	1893
135	Dr D. Menzies, 20 Rutland Square,	1878
	Dr A. Miles, 1 George Square,	1892
	Dr J. Mill, 26 N. Fort Street, Leith,	1886
	A. G. Miller, Esq., 7 Coates Crescent, *President*,	1867
	Dr W. H. Miller, 51 Northumberland Street,	1895
140	Dr D. Milligan, 11 Palmerston Place,	1885
	Sir Arthur Mitchell, 34 Drummond Place,	1859
	Dr James Mowat, 26 Minto Street,	1893
	Dr John Mowat, 6 Buccleuch Place,	1885
	Dr Claud Muirhead, 30 Charlotte Square,	1866
145	Dr R. Milne Murray, 11 Chester Street,	1884
	Dr John Orr, 1 Rillbank Crescent,	1895
	Dr W. Basil Orr, 13 Braid Road,	1893
	Dr G. Keppie Paterson, 19 Albany Street,	1889
	Dr D. Noël Paton, 22 Lynedoch Place,	1885
150	Dr H. A. Peddie, 24 Palmerston Place,	1893
	Dr R. W. Philip, 45 Charlotte Square,	1883
	Dr Playfair, 5 Melville Crescent,	1874
	Dr T. Proudfoot, 30 Lauriston Place,	1889
	Dr R. B. Purves, 17 Walker Street,	1899
155	Dr Harry Rainy, 25 George Square,	1896
	Dr Rattray, Portobello,	1874
	Dr James Ritchie, 22 Charlotte Square,	1873
	Dr R. Peel Ritchie, 1 Melville Crescent,	1862
	Dr Argyll Robertson, 18 Charlotte Square,	1861
160	Dr G. M. Robertson, The Copse, Larbert,	1890
	Dr William F. Robertson, 20 Dalrymple Crescent,	1893
	Dr W. G. Aitchison Robertson, 26 Minto Street,	1892
	Dr R. M. Ronaldson, 17 Morningside Road,	1895
	Dr T. R. Ronaldson, 8 Charlotte Square,	1877

		Date of Admission.
165	Dr William Russell, 3 Walker Street,	1884
	Dr E. G. Salt, 50 George Square,	1895
	Dr James Scott, 43 Minto Street,	1895
	Dr Andrew Semple, 10 Forres Street,	1883
	Dr Theodore Shennan, 35 Mayfield Gardens,	1897
170	Sir John Sibbald, 18 Great King Street,	1859
	Professor Simpson, 52 Queen Street,	1859
	Dr A. A. Scot Skirving, 29 Drummond Place,	1897
	Dr A. T. Sloan, 22 Forth Street,	1885
	Dr Andrew Smart, 15 Rutland Square,	1865
175	Dr G. D. Smith, 148 Ferry Road,	1877
	Dr James Smith, 1 Parson's Green Terrace,	1891
	Dr John Smith, 11 Wemyss Place,	1856
	Dr J. Cormack Smith, 9 Brunton Place,	1896
	Dr William Smith, 14 Hartington Gardens,	1890
180	Dr John Stevens, 2 Shandon Street,	1892
	Dr J. S. Stewart, 15 Merchiston Place,	1885
	Dr Robert Stewart, 42 George Square,	1892
	Dr William Stewart, 146 Ferry Road, Leith,	1889
	H. J. Stiles, Esq., 8 St Colme Street,	1889
185	Dr S. Stirling, 4 Coates Crescent,	1891
	Dr A. Stodart-Walker, 30 Walker Street,	1894
	Dr John Strachan, Dollar,	1867
	Dr Allan C. Sym, 144 Morningside Road,	1889
	Dr William G. Sym, 20 Alva Street,	1889
190	Dr W. Taylor, 12 Melville Street,	1871
	Dr C. C. Teacher, 8 Nile Grove,	1887
	Dr C. H. Thatcher, 8 Melville Crescent,	1876
	Dr R. Thin, 38 Albany Street,	1890
	Dr Alexis Thomson, 32 Rutland Square,	1887
195	Dr John Thomson, 14 Coates Crescent,	1887
	Dr T. J. Thyne, 2 Dean Terrace,	1898
	Dr John Tod, 93 Ferry Road, Leith,	1895
	Sir John Batty Tuke, M.P., 20 Charlotte Square,	1864
	Dr F. J. Turnbull, 6 Randolph Place,	1896
200	Dr Dawson Turner, 37 George Square,	1890
	Dr Logan Turner, 20 Coates Crescent,	1892
	Dr Underhill, 8 Coates Crescent,	1872
	Dr Norman Walker, 7 Manor Place,	1891
	D. Wallace, Esq., 11 Rutland Street,	1887
205	Dr Douglas C. Watson, 22 Coates Crescent	1894
	Dr P. H. Watson, 16 Charlotte Square,	1856
	Dr N. P. Watt, 1 Denham Green Terrace, Trinity,	1898
	Dr A. D. Webster, Belleville Lodge, Blacket Avenue,	1883
	J. L. Wilson, Esq., 4 Buccleuch Place,	1883
210	Dr T. D. Wilson, 10 Newington Road,	1880
	Dr Russell E. Wood, 9 Darnaway Street,	1883
	Dr W. Fraser Wright, Bonnington Mount, Bonnington Ter.,	1894
	Professor John Wyllie, 44 Charlotte Square,	1868
	Dr P. A. Young, 25 Manor Place, *Vice-President*,	1870
215	Dr R. J. Erskine Young, 8 Alva Street,	1892

(*b.*) Members Exempted under Rule V. from paying the Annual Subscription.

		Date of Admission.
	Dr D. Aitken, 152 *Packington Street, Islington, London, N.*,	1887
	Dr D. H. Anderson, 131 *Abbey Road, Barrow-in-Furness*,	1887
	Dr Archibald, *Hazelden, Wimbourne Road, Bournemouth*,	1882
	Dr J. A. Armitage, 28 *Waterloo Road South, Wolverhampton*,	1887
220	Dr W. Badger, *Penicuik*,	1882
	Dr Edwin Baily, *Oban*,	1883
	Dr Andrew Balfour, *Portobello*,	1874
	Dr James Craig Balfour, *Westfield, Colinton*,	1884
	Dr J. H. Balfour, *Portobello*,	1881
225	Dr Alexander Ballantyne, *Dalkeith*,	1872
	Dr Harold S. Ballantyne, *Dalkeith*,	1899
	Dr W. H. Barrett, 29 *Park Crescent, Hesketh Park, Southport*,	1890
	Dr George T. Beatson, 7 *Woodside Crescent, Glasgow*,	1895
	Surgeon-Captain C. H. Bedford, *Bengal Army, care of W. Watson & Co.*, 28 *Apollo Street, Bombay*,	1889
230	Dr Benjamin D. C. Bell, *Kirkwall*,	1889
	Dr G. J. H. Bell, *Bengal Army*,	1884
	Dr G. H. Bentley, *Kirkliston*,	1877
	Dr J. S. Beveridge, *Melton Constable, Norfolk*,	1861
	Dr J. W. Black, 15 *Clarges Street, Piccadilly, London, W.*,	1865
235	Dr P. G. Borrowman, *Viewfield, Crieff*,	1893
	Dr D. G. Braidwood, *Halkirk, Caithness*,	1889
	Dr David Brodie, 68 *Hamilton Road, Highbury Park, London*,	1865
	Dr Andrew Brown, 1 *Bartholomew Road, Kentish Town, London, N.W.*,	1884
	Dr John Brown, *Stockbridge House, Padiham, Lancashire*,	1878
240	Dr J. Macdonald Brown, 5 *Lymington Road, West Hampstead, London, N.W.*,	1883
	Dr S. W. Carruthers, 44 *Central Hill, Norwood, London, S.E.*,	1899
	Dr R. D. Clarkson, *Falkirk*,	1896
	Professor Cleland, *The University, Glasgow*,	1864
	Dr A. R. Coldstream, 24 *Lung Arno Nuovo, Florence, Italy*,	1878
245	Dr John Connel, *Peebles*,	1876
	Dr William Craig, *Cowdenbeath, Fife*,	1894
	Dr J. R. Crease, 2 *Ogle Terrace, South Shields*,	1885
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	Dr P. M. Deas, *Wonford House, Exeter*,	1868
250	Dr Archibald Dickson, *Hartree House, Biggar*,	1871
	Dr A. Halliday Douglas, *Haddington*,	1842
	Dr A. Home Douglas, 5 *Rue de Russie, Nice, France*,	1889
	Dr J. J. Douglas, 42 *Centrall Hill, Upper Norwood, London*,	1891
	* Dr W. B. Dow, *Dunfermline*,	1879
255	Dr Thomas Easton,	1894
	Dr J. W. Eastwood, *Dinsdale Park, Darlington*,	1871
	Dr F. A. Elkins, *The Asylum, Leavesden, near King's Langley*,	1893
	Dr R. W. Felkin, 6 *Crouchhall Road, London, N.*,	1885
	Sir R. B. Finlay, Q.C., M.P., *Middle Temple, London*,	1864
260	Dr Simson C. Fowler, *Juniper Green*,	1892
	Dr F. W. Foxcroft, 33 *Paradise Street, Birmingham*,	1893
	Dr Dyce Fraser, 1 *Oxford Villas, Teddington*,	1883
	Dr J. Hosack Fraser, *Fernfield, Bridge of Allan*,	1895
	Professor Gamgee, 8 *Avenue de la Garve, Lausanne, Switzerland*,	1863

		Date of Admission.
265	Dr William Gayton, *Bartram Lodge, Fleet Road, Hampstead, London, N.W.*,	1886
	Dr F. M. Graham, *Willowbridge, Market Drayton*,	1894
	Dr W. C. Greig, *Otago, New Zealand*,	1884
	Dr Groesbeck, *Cincinnati*,	1875
	Dr W. B. T. Gubbin, *Redland Grove, Bristol*,	1893
270	Dr John Haddon, *Denholm, Roxburghshire*,	1883
	Dr Archibald Hall, *Montreal*,	1853
	Professor D. J. Hamilton, *The University, Aberdeen*,	1876
	Dr J. W. Hamp, *Penn Road, Wolverhampton*,	1887
	Dr John Hardie, *Stogursey, near Bridgewater*,	1895
275	Dr A. W. Hare, *Newquay, Cornwall*,	1883
	Dr J. Home-Hay, *Alloa*,	1880
	Professor J. Berry Haycraft, 1 *St Andrew's Place, Cardiff*,	1889
	Dr Stanley Haynes, *Malvern, Worcestershire*,	1864
	Dr R. Dundas Helm, 3 *Alfred Street N., Portland Square, Carlisle*,	1892
280	Dr R. E. Horsley, *Jud-Falls, Stoneyhurst, Lancashire*,	1886
	Professor A. W. Hughes, *King's College, London*,	1889
	Dr James Hunter, *St Catherine's, Linlithgow*,	1890
	Dr W. Hunter, 103 *Harley Street, Cavendish Square, London, W.*,	1887
	Dr David Huskie, *Moffat*,	1899
285	Dr J. Carlyle Johnstone, *The Asylum, Melrose*,	1882
	Dr D. W. Johnston, *P.O. Box* 2022, *Johannesburg, South Africa*,	1893
	Dr James Johnston, 53 *Princes Square, Bayswater, London, W.*,	1871
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290	Dr George Keith, *Moidart Cottage, Currie*,	1845
	Dr Skene Keith, 42 *Charles Street, Berkeley Square, London, W.*,	1885
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	Professor R. F. C. Leith, M.B., *Mason University College, Birmingham*,	1886
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295	Dr Robert Lucas, *Dalkeith*,	1875
	Dr Donald Macaulay, *Jumpers' Gold Mining Co., P.O. Box* 1043, *Johannesburg*,	1895
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	Dr K. N. Macdonald, 21 *Clarendon Crescent*,	1880
	Dr W. B. Macdonald, *Dunbar*,	1888
300	Dr John A. Macdougall, *Cannes, France*,	1875
	Professor J. M'Fadyean, 101 *Great Russell Street, London, W.C.*,	1888
	Dr F. Reid Mackay, 27 *York Place, Harrogate*,	1899
	Dr John Mackay, *Aberfeldy*,	1881
	Dr W. B. Mackay, 23 *Castlegate, Berwick-on-Tweed*,	1893
305	Professor M'Kendrick, *The University, Glasgow*,	1870
	Dr James Mackenzie, 68 *Bank Parade, Burnley, Lancashire*,	1894
	Dr T. J. Maclagan, 9 *Cadogan Place, Belgrave Square, London, S.W.*,	1875
	Dr Roderick M'Laren, 23 *Portland Square, Carlisle*,	1882
	Dr S. Macvie, *Chirnside*,	1897
310	Dr J. W. Martin, *Burnfoot, Cluden, Dumfries*,	1888
	Dr A. Matthew, *Corstorphine*,	1882
	Dr Andrew W. Messer, *Lenington, Scotswood-on-Tyne*,	1899
	Dr James Middlemass, *Boro' Asylum, Ryhope, Sunderland*,	1895
	Dr E. A. Mills-Roberts, *Bryn Meurig, Bethesda, Bangor*,	1897
315	Dr J. Moolman, *Cape of Good Hope*,	1877

		Date of Admission.
	Dr E. C. Moore,	1892
	Dr A. E. Morison, *Wellington Road, West Hartlepool,*	1889
	Dr J. Rutherford Morison, 14 *Saville Row, Newcastle-on-Tyne,*	1882
	Professor Robert Muir, *The University, Glasgow,*	1894
320	Dr D. G. Macleod Munro, 1 *Oriel Villas, Cheltenham,*	1898
	Dr J. Ivor Murray, 2 *Pavilion Square, Scarboro',*	1857
	Dr Andrew Scott Myrtle, *Harrogate,*	1859
	Dr Leith Napier, *The General Hospital, Adelaide, South Australia,*	1879
	Dr T. Goodall Nasmyth, *Cupar-Fife,*	1884
325	Dr Ernest F. Neve, *Srinagar, Kashmir, India,*	1884
	Dr Peter Orphoot, *Greenhythe, North Berwick,*	1865
	Dr T. Wyld Pairman, *H.M. Prison, Lyttelton, New Zealand,*	1884
	Professor Bell Pettigrew, *St Andrews,*	1873
	Dr Alexander Peyer, *Zürich,*	1893
330	Dr J. A. Philip, *Rue Victor Hugo, Boulogne-Sur-Mer, France,*	1878
	Professor W. S. Playfair, 38 *Grosvenor Street, London, W.,*	1857
	Dr J. H. Pringle, 172 *Bath Street, Glasgow,*	1886
	Dr S. Hale Puckle, *Bishop's Castle,*	1885
	Dr J. Maxwell Ross, *Avenel, Maxwelltown, Dumfries,*	1882
335	Dr J. R. Home Ross, *Burmah,*	1888
	Dr Joseph C. Ross, *Withington,*	1884
	Dr S. Rumboll, *Cavendish House, Leeds,*	1887
	Dr Thomas Russell, 27A *Westmuir Street, Parkhead, Glasgow,*	1888
	Dr Gordon Sanders, *Cannes, France,*	1892
340	Dr F. A. Saunders, *Grahamstown, South Africa,*	1884
	Dr Thomas R. Scott, *Musselburgh,*	1884
	Dr C. A. E. Sheaf, *Ovalu, Fiji,*	1871
	Dr G. F. Shiels, 229 *George Street, San Francisco,*	1887
	Dr W. H. Shirreff, *Port Fairy, Victoria, Australia,*	1883
345	Dr William Simmers, *Denburn, Crail,*	1894
	Dr T. Skinner, 6 *York Place, Portman Square, London, W.,*	1856
	Dr John Smith, *Brycehall, Kirkcaldy,*	1889
	Dr W. Ramsay Smith, *The General Hospital, Adelaide, South Australia,*	1892
	Dr Van Someren, *Northwood, Middlesex,*	1845
350	Dr Somerville, *Galashiels,*	1877
	Dr A. M. Stalker, 140 *Nethergate, Dundee,*	1893
	Dr Graham Steell, 96 *Moseley Street, Manchester,*	1877
	Professor Stephenson, *University, Aberdeen,*	1861
	Dr Robert Stirling, 4 *Atholl Place, Perth,*	1891
355	Professor Ralph Stockman, *The University, Glasgow,*	1891
	Dr H. R. Storer, *Newport, Rhode Island, U.S.,*	1855
	Dr John Struthers, *Ngamakwe, Transkei, South Africa,*	1895
	Dr C. A. Sturrock, *Dunfermline,*	1894
	Dr J. F. Sturrock, *Arima, Broughty Ferry,*	1887
360	Professor J. Symington, *Queen's College, Belfast,*	1878
	Dr C. Templeman, *Sanitary Office, Bell Street, Dundee,*	1891
	Dr Thin, 63 *Harley Street, Cavendish Square, London, W.,*	1861
	Dr Alexander Thom, *Viewfield, Crieff,*	1884
	Dr J. Stitt Thomson, *The Mount, Lincoln,*	1877
365	Dr J. Batty Tuke, jr., *Balgreen, Murrayfield,*	1886
	Dr R. S. Turner, *Keith,*	1867
	Dr T. Edgar Underhill, *Dunedin, Burnt Green, Worcestershire,*	1885
	Dr A. R. Urquhart, *Murray House, Perth,*	1893
	Dr Gopal Govind Vatve, *care of H.H. the Rajah of Miraj, Bombay, India,*	1895
370	Dr A. B. Whitton, *Aberchirder,*	1886
	Dr George R. Wilson, *Mavisbank, Polton,*	1892

		Date of Admission.
	Dr Oswald G. Wood, *Indian Army*, ...	1886
	Professor G. Sims Woodhead, *The University, Cambridge*, .	1883
	Dr Yellowlees, *Gartnavel Asylum, Glasgow*, . . .	1862
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TRANSACTIONS

OF THE

MEDICO-CHIRURGICAL SOCIETY OF EDINBURGH

FOR SESSION LXXIX, 1899-1900

Meeting I.—November 1st, 1899

SIR JOHN BATTY TUKE, *President, in the Chair*

I. ELECTION OF OFFICE-BEARERS

The following gentlemen were elected office-bearers:—*President*, A. Gordon Miller, Esq.; *Vice-Presidents*, Dr P. A. Young, Dr James Dunsmure, and Dr James Andrew; *Council*, Drs H. Melville Dunlop, George Mackay, H. Alexis Thomson, George L. Gulland, Sir John Batty Tuke, J. J. Graham Brown, R. M'Kenzie Johnston, and David Wallace; *Treasurer*, Dr Harvey Littlejohn; *Secretaries*, Mr J. M. Cotterill and Dr H. Melville Dunlop; *Editor of Transactions*, Dr William Craig.

II. ELECTION OF MEMBER

Henry A. Leebody, M.B., C.M., Corstorphine, was elected an Ordinary Member of the Society.

III. EXHIBITION OF PATIENTS

1. *Dr Alexander James* exhibited (*a*) a case of slight CRETINISM. The patient was a boy, aged 7, and he presented very well many of the ordinary aspects of the disease—the

type of face, the open mouth, the projecting tongue, the large abdomen and protruding umbilicus. The limbs were not crooked, but the growth was stunted, as evidenced by the fact that he was four inches shorter than his sister who was two years younger than himself. The thyroid was absent, and mentally he was deficient. A specially noteworthy feature was that his sister was affected with a well-marked goitre. Under the thyroid treatment he was improving rapidly, both physically and mentally.

(*b*) An example of a RARE SKIN AFFECTION. The subject of this was a man aged 25, and Dr James had to thank Dr Allan Jamieson for having given him references to examples of a similar disease. The affection was that known as Keratodermia Essentialis. The disease had existed for some three weeks, and an interesting feature was that some seven years previously the patient had passed through what appeared to be a very similar attack, having been ill at that time for nine weeks.

2. *Dr F. D. Boyd* exhibited a child exemplifying a CRETINOID CONDITION. The patient, a little girl aged 14 years, showed marked developmental deficiency, both physically and mentally. While 14 years old her height was 4 ft., her weight 3 st. 6½ lb. Intellectually she was far behind her age, being only in the first standard.

Shortly after birth the parents noticed that there was some swelling of the neck which had since increased. The swelling was the result of an enlargement of the thyroid gland, both lobes being very markedly enlarged and apparently of a fibrous nature with possibly some cystic formation. The child's expression was peculiar, the eyes being prominent and the bridge of the nose flattened. This might partly be accounted for by the presence of adenoid growth in the naso-pharynx which had been recently removed. There was no thickening of the lips, enlargement of the tongue, fulness of the abdomen, crooking of the legs or roughening of the skin.

IV. Original Communications

1. VALEDICTORY ADDRESS

By Sir John Batty Tuke, M.D., D.Sc., F.R.C.P.Ed., President

THE only onerous or difficult duty imposed on the President of the Medico-Chirurgical Society is the delivery of a valedictory address. From the excellent management of the council and secretaries all the work of the Society goes on smoothly; hitches never seem to arise or difficulties to occur—if they do, they are smoothed over by the diplomacy of these officers. Due to the energy of the editor, the *Transactions* appear with unerring punctuality, admirably arranged, and thoughtfully classified. And, as the result of the highly courteous tone which characterises the discussions, the president's function as moderator is uniformly pleasant in its exercise, inasmuch as the utmost good fellowship pervades even the most animated discussions.

Before reviewing the work of the last two sessions I have to thank the Society most heartily and sincerely for having elected me to this most honourable and comfortable office. Curiously enough this is the first opportunity I have had of acknowledging this compliment, for on the occasions of election and re-election I was compelled by official duties elsewhere to be absent from the meetings at which I should have expressed my sense of gratitude. Whilst doing so let me add that I deeply regret that the duties of the chair have been performed in a perfunctory manner. I have said that the prescribed duties are easy; but when I look back on the energy which my predecessors threw into their work, I recognise that the mere occupancy of this chair does not constitute the sole duty of the president. I can only plead long and serious illness, and the weight of advancing years, as excuses for my shortcomings.

The preparation of a valedictory address is, I suspect, in the majority of cases, not such an easy or agreeable task as the composition of an inaugural address. In the latter case one has the broad future before one, and the sentiment of Hope is more present in the mind; *illa quidem fallax, sed tamen apta dea est:* in the former one has to deal with accomplished facts;

and it almost inevitably happens that their consideration gives rise to mingled feelings of regret and satisfaction. In the retrospect of the last two years these feelings assert themselves strongly: satisfaction connected with the good work the Society has done, regret at the loss of members who in bygone years did much to support and enhance its efficiency.

During this period we have lost by death thirteen members, and amongst that number several men of mark. I do not propose to dwell long on this subject, but I am sure you would not wish that I should pass over in silence the names of men with whom the Society has been more closely associated, or omit to express our deep regret at their loss.

From the list of honorary members has dropped the name of William Jenner, a pillar of medicine, and of whom it has been said (and I believe with strict accuracy), that he never made a mistake in matters connected with professional ethics.

When John Struthers, corresponding member, died, we lost a strong man. He made the University of Aberdeen an important medical school in the face of many difficulties. This, as in fact all of his works, he effected by steady determination; he was indefatigable in everything he took in hand; and, although as regards certain of the objects he had in view there was not perfect unanimity amongst his fellows, no one doubted the purity of his purpose. He did much useful work for his time and generation.

From amongst the ordinary members we have lost the venerable John Moir, who joined the Society in 1836; James Cappie, a philosophic, ingenious and gentle man; William Tasker Lundie, a rising practitioner of great promise; Alexander Moir, Lawson Tait and Sinclair Coghill, all closely connected with the Edinburgh School.

It is needless to speak in this hall of William Rutherford as a physiologist and teacher, for as such you all knew him; but I doubt whether many knew what a kind, generous spirit was hidden under a somewhat cold manner. He never let his left hand know what his right was doing; every now and then, in some roundabout way, those who knew him best heard of acts of liberality for which he never sought or got credit. Nothing softened him so much as a tale of distress, and I take it that, if the gentleness of his nature was thus manifested, it

extended itself to many other relations of life. In point of fact I know it did.

With John Duncan died a strong outstanding personality. It was not his professional attainments, great though they were, that gained for him pre-eminence amongst us so much as the pure judicial quality of his mind, the independence of his character, and the manliness of his disposition. These qualities, carried into the conduct of business of whatever kind, made him the trusted adviser of all sorts and conditions of men, whether they were politicians, financiers, members of his own profession, or dependents. His old gamekeeper wrote when he heard of his death : " He was a noble gentleman, and the kindest of masters." To my mind, Duncan's walk and conversation is well expressed in the words of a great living poet :—

> " So walked he from his birth,
> In simpleness, and gentleness, and honour and clean mirth."

The material prosperity of the Society continues unabated. Notwithstanding the heavy death roll the number of ordinary members stands at 373 ; the total membership amounts to 416. There are vacancies in the lists of honorary and corresponding members, and it will be your duty to consider on whom you will confer this honourable distinction. The treasurer informs the council that the report he will submit to you in due course will be satisfactory. We all regret that Dr Mackenzie Johnstone feels compelled to resign the treasurership, as also that Dr Graham Brown demits office as secretary. I shall ask you later on to accord a special vote of thanks to these gentlemen for the admirable manner in which they have performed their duties.

Even a cursory review of the work of the last two sessions must convince any unprejudiced mind that the scientific spirit pervades the Edinburgh School of Medicine and Surgery very thoroughly. In every paper that has been submitted to the Society there is ample evidence of careful observation, deep thought and deliberation. New facts and theories have been applied to the study of almost every class of disease, and to that of individual cases. The discussions have been animated, and at large meetings the members have expressed their opinions on the various theses freely and fairly, whether those opinions were adverse to or in accordance with the views of the

author. The exhibition of patients, pathological specimens, and instruments has, as usual, constituted a large part of the work, and the special clinical meetings have been markedly successful. Like the previous occupant of this chair, Dr Argyll Robertson, I must express a strong desire that some arrangement could be come to by which the two departments of the Society's work should not interfere one with the other. I do so in the interest of future presidents. Perhaps it is only those who have filled that office who can realise how painful it is to cut short a debate or restrict discussion in order that the agenda on the billet should be overcome. It has frequently happened that, owing to the accommodation of exhibits, the reading of important papers has been delayed to such a late hour that their full consideration had been impossible. This appears to me to be hardly fair to men who have been engaged in the preparation of their dissertations for weeks or months, and for whom it may be necessary to present them at a special date. The council has considered the matter, and has instructed the secretaries either to call an extra clinical meeting, or to arrange at least one of the ordinary meetings so that the time may be equally occupied by exhibits and papers.

Many of the papers submitted to the Society have been of great importance and interest. The study of the Pathology of a Fit of Gout by Dr George Balfour was characterised by the learning, extended knowledge of medical literature, and elegance of diction for which he has gained so high a reputation. It illustrated the truth of Cullen's position—that to render the association of practice and theory safe it is necessary to cultivate theory to its fullest extent. I regret that circumstances closely connected with the subject dealt with prevented me from hearing the paper read and discussed. Great interest attached to the paper on the Etiology of Gout by Dr Chalmers Watson, who, it is understood, is continuing investigations which must tend to increase our knowledge of the subject. Dr Claude Ker's consideration of the Serum Diagnosis of Enteric Fever was most welcome, and his description, written in association with Dr Harvey Littlejohn, of "An Outbreak of Typhus Fever," must always hold an important place in the history of that disease. Dr James' contributions on Syphilitic Osteitis, Tubercular Neuritis, and Liver Cirrhosis were illustrations of the careful and persevering methods of investigation so con-

sistently followed by that very original thinker. Dr Francis Boyd presented a singularly interesting communication on Regurgitation of the Pulmonary Artery, which should lead up to thorough discussion; this, however, was impossible in consequence of the late hour at which the paper was read. The papers on the Acute Pneumonia of Children, by Dr James Carmichael; on Yellow Atrophy of the Liver, by Dr Dobie; on Rheumatic Myositis and on the Etiology and Treatment of Glandular Enlargements of the Neck, by Mr Miller; and on the Treatment of Ozæna, by Dr M'Bride, aroused the interest of the Society as shown by the full criticism to which they were submitted. To Dr Allan Jamieson we are indebted for elaborate descriptions and illustrations of various forms of skin disease; to Mr Wallace for his paper on Movable Kidney; to Mr Alexis Thomson for his communication on the treatment of Enlarged Prostate; and to Mr Cathcart for notes on Peritonitis as a cause of increased peristalsis in the rectum. Dr Lovell Gulland in his paper on Sulphonal Poisoning, collated all that was known of the subject, with the phenomena presented by a case which had come under his observation. Under the modest title of "Some Observations on the Development of the Testicle," Dr Foulis submitted a dissertation, founded on a prolonged and laborious investigation which will doubtless exercise important bearings on accepted views. We have been specially rich in dissertations on diseases of the nervous system. In certain instances the physician and surgeon, or the physician and pathologist, have collaborated in producing works which extend the horizon of our knowledge on the localisation of cerebral function, and of methods by which morbid conditions of the organ may be combated. The conclusions which Dr Bruce laid before the Society in his paper "On the Localisation and Symptoms of Disease of the Cerebellum in relation to its Anatomical Connections," in what appeared to me a somewhat tentative and reserved manner, have met with confirmation by subsequent clinical experience. He is to be congratulated on this rapid realisation of the scheme propounded before the Society, which serves to confirm him in the distinguished position he holds amongst neurologists. The contributions of Mr Miles, of Dr Shennan, and of Mr Cotterill have all important bearings on the diagnosis and treatment of intra-cranial disease. Finally we must all recognise

the high value of Dr John Thomson's lecture on the Diagnosis and Prognosis of certain forms of Imbecility in Childhood, which was followed by a brilliant lantern demonstration.

It is somewhat remarkable that not one of the twenty-six dissertations submitted to the Society has touched on those medical matters which have exercised the public mind during the last two years. I allude specially to the vaccination question, the prevention of the spread of syphilis, and the arrest of tubercular disease. I believe that as regards the first the profession feel that it has said its say, finding the incontrovertible evidence it had produced of the necessity for strengthening the Acts set aside for mere party purposes, and finding its representatives in Parliament giving themselves and the position away, thought it wiser to let things swing. Perhaps it was wise so to do. I do not suppose you are all salmon fishers, so let me illustrate the position by reference to a manœuvre occasionally practised by the professors of that art. When you have hooked a big fish in a strong stream, and the fish will work down to a place where disaster is inevitable, it is a good plan suddenly to give him a few yards of line, when, believing himself to be free, he turns upwards, and you can guide him into calmer waters. This is what seems to have happened in certain centres of agitation, where we are informed that the conscientious objector is celebrating his victory by getting himself and his children vaccinated. It is one of those instances of paradoxical rightness which occasionally follows on reduction of strain ; or, is it that the good sense of the nation is at last asserting itself ?

Although the Society took no part in the vaccination question, it might be well were it to consider the propriety of taking action as regards the popular interest in tuberculosis and specific disease. It is interesting to compare the indifference with which the public relinquished the Contagious Diseases Acts with the avidity with which it seized on proposals to deal with tuberculosis on general hygienic principles. Of course the reasons for this are not far to seek. The details of venereal disease are revolting, they must be spoken of with bated breath, and curious though not unnatural sympathies were aroused in connection with the means considered necessary for reducing its ravages. With tuberculosis, on the other hand, scandal is not associated, and its natural history can be discussed *coram*

publico. The general recognition of the fact that a disease which makes itself so forcibly, frequently, painfully and openly felt, is communicable, and that communication may be preventable, has created a special and widespread interest. That it is the duty of the medical profession to maintain that interest no one will gainsay, but the best means of so doing is to express to the public opinions bearing not so much as to what could possibly be done under an ideal state of social existence, but as to what can be done under existing conditions, and so to lead gradually upwards from present imperfections to a nearer approach to the ideal. The public must be taught not to expect too much, it must be educated to a degree of knowledge which will enable it to adopt or refuse, on a rational understanding of the questions involved, measures, which however expedient for the common good, might complicate social relations. The community must be made to understand that, before any material decrease of tubercular infection can be expected, it has to face, amongst others, such serious social questions as compulsory notification and isolation. It has done so as regards other forms of disease, and, unless a new class of faddist arises, it may adopt important precautionary measures as regards tuberculosis. The Town Council of Edinburgh has taken up the consideration of the question, and has consulted the Royal Colleges, who have reported very fully. Under these circumstances it might be a work of supererogation for this Society to supplement this report at present; still, when the Town Council formulates a scheme it should, in my opinion, be carefully considered by us, and the result of our deliberation should be forwarded to the municipal authorities. It would be of importance to the public to place on record the opinion of a body which comprises practitioners of medicine and surgery distributed over the country; and I think I am justified in adding, that it is to the interest of the Society to make itself felt in all deliberations dealing with the health of the community.

2. ON THE IMPORTANCE OF EXAMINING THE BLOOD IN DOUBTFUL CASES OF DISEASE

By G. LOVELL GULLAND, M.A., B.Sc., M.D., F.R.C.P.Ed., Assistant Physician to the Royal Infirmary, Physician to the Victoria Hospital for Consumption, etc., Edinburgh

I WISH at the outset to premise that I do not intend to speak specially about the examination of the blood in such diseases as pernicious anæmia or chlorosis, for in these and similar conditions everyone is agreed that the blood *ought* to be examined, and such examinations are not infrequently actually made, even though the results are not always scientifically accurate. But there is, in this country in particular, a general and widespread ignorance of the amount of information which can be gained from the thorough examination of the blood in general diseases, apart from the diseases of the blood-forming organs, and of the valuable indications for diagnosis, prognosis, and treatment which can be obtained in this way. I say in this country advisedly, for the vast bulk of hæmatological knowledge is derived from observations made either in the German-speaking countries or in the United States. One reason for this may be that we have not here such diseases as malaria to draw our attention to the blood in obscure conditions. There must, however, in addition be some fault in our education, for certainly not one in a hundred of our young graduates knows how to conduct a blood examination properly, or, which is even more important, how to interpret his results when he has got them; and if he has the knowledge, it is too often forgotten or neglected when he goes into practice. If students were less vexed with the skeleton of the crocodile, and with the phenomena of electrotonus, there might be time for them in their course to learn matters of value to them, and there would be some chance of medicine becoming a scientific profession instead of a business.

There is in the minds of some men a positive dread of making blood examinations. For example, I came across an instance not long ago of a practitioner who had to examine a case for life insurance. The proposer had an enlarged spleen, and the examiner suspected that he might have leucocythæmia. Instead of pricking the finger and making sure, as he might have done in ten minutes, the doctor gravely wrote on the form

that "of course he had not examined the blood, as there was a considerable danger of erysipelas from the necessary puncture, and he feared to involve the company and himself in a lawsuit for damages if this happened!" In all the hundreds of punctures that I have made, and the thousands I have read of, I never heard of the slightest ill effect from the necessary needle-prick. A somewhat similar cautiousness on the part of a colleague once put me in some embarrassment. There was a question in a case whether the symptoms were due to pernicious anæmia or to a hidden gastric cancer. The case had been seen by a consultant very much senior to myself, who not only had made no blood examination, but had so impressed on the patient the danger of his losing a single drop of blood, that I had great difficulty in persuading him to let me examine it. The case, when examined, turned out to be a pernicious anæmia of the chronic variety, and did well on appropriate medication, which was determined by the blood examination. On the other side, there is the man who expects you to diagnose pernicious anæmia without examining the patient and without counting his corpuscles, by the aid of two dirty coverglasses which have had a drop of blood squeezed between them, and which he has not always taken the pains to draw apart!

Perhaps the best way to press the importance of the matter is to point out that blood is the only *tissue* of the body which we have the opportunity of examining microscopically and chemically during life, and that in most other cases we can only examine tissue products. What would we not give to be able to examine a portion of the kidney in a doubtful case of albuminuria, for instance? Further, while there are special diseases which affect mainly the blood, there is probably no disease which does not affect the blood-mixture, as the Germans call it, in some way. Our present methods are often too rough to enable us to seize the exact difference; in most cases we can only trace comparatively coarse morphological changes, but there is no reason to believe that the discovery of serum diagnosis in typhoid fever and other infectious diseases has exhausted the possibilities in that direction. We may hope to find subtle changes in the diseases of metabolism, for instance, and there are many avenues of work whose openings we can see, but not their ends.

Leaving out of sight, however, for the time all these finer

points, in an ordinary blood examination we have to look to the number and condition of the red corpuscles and their content of hæmoglobin; the number of the white corpuscles, and the percentage of the different varieties present; the number of blood plates, and the rapidity of formation of the fibrin network. In special cases, of course, such things as the plasmodium malariæ or the presence of organisms may have to be looked for, but these I need not now further allude to.

First as to the number and condition of the red corpuscles. These points are of importance in the diagnosis from primary blood diseases, but apart from that they are of comparatively little service in making the diagnosis of the disease which is present, except in so far as they enable us to discriminate between a grave illness and a slight one; for the reason that the conditions which produce anæmia are almost infinite in number, and any two causes of equal severity will produce just the same kind of anæmia, no matter how widely diverse they may be. For example, gastric cancer, bleeding piles, and septicæmia will produce anæmias which, as far as the red cells go, are indistinguishable from one another. The condition of the red cells is invaluable on the other hand in helping us to estimate the severity of the individual case and its prognosis, the amount of recuperative power, and, of course, to trace progress. It is obvious that a patient suffering from, say pneumonia or phthisis, has a much better chance if he starts with 5,000,000 corpuscles to the cubic millimetre than if he has only 2,500,000, and those small and misshapen.

The percentage of hæmoglobin gives very much the same kind of information as the red cells; in many cases the amount of loss is proportional to the loss of red cells, and in many others—where the corpuscles are small and of light weight—the hæmoglobin is proportionally diminished. In one case only, that of pernicious anæmia, the hæmoglobin is relatively increased.

The number of blood plates is difficult to estimate, and changes do not usually give us any information that cannot be got in other ways. The same may be said with regard to the rapidity of fibrin formation; the one point in which we may get special help from it, is in making a diagnosis between suppuration and malignant disease. In the former the fibrin

is increased, in the latter it remains unaltered, though the blood in other respects may be very similar.

But it is the white corpuscles which help us most. An increase of them, leucocytosis; a diminution, leucopenia; a normal number, and almost as much the proportion of the different varieties which is present, and in some cases the presence or absence of varieties which are not usually found in the blood, may all give us important information, not only for diagnosis, but also for prognosis.

I can best illustrate the use which is to be made of the information gained by blood examinations by recounting briefly some of the many cases in which, either in my own practice, in consultation, or by the kindness of my colleagues, I have examined the blood for diagnostic purposes, and by pointing out the method of interpretation in each case, which, as in other methods of diagnosis, usually involves the weighing of *pros* and *cons*.

A case which made a deep impression on me at the time, was that of a young man on whom, on the 1st of the month, a cosmetic operation was performed. Everything went well until the evening of the 5th, when the temperature rose to 104°, and the patient developed a sore throat. The wound seemed to be all right, there was no pain; the patient was obviously very ill, but there was nothing on which a definite diagnosis could be founded, and the surgeon was naturally unwilling to believe that it was a septicæmia. The question put to me was, is this influenza (which was epidemic at the time), tonsillitis, or septicæmia? On the 7th I saw the case, when the temperature was about 103°, and examined the blood. The red corpuscles numbered 2,000,000 in the cubic millimetre, with about 30 per cent. of hæmoglobin, and the whites numbered 24,200, and were almost all polymorphonuclear. On the strength of this I assured the surgeon that it was septicæmia, and advised the opening up of the wound and the injection of antistreptococcic serum. Nothing was found in the wound, the temperature fell to 99·5 after the injection of the serum, but rose again and remained about 104°, in spite of a second injection, until death, which took place on the 9th. The interpretation of the blood examination was as follows:—Influenza was at once excluded by the leucocytosis, for that disease does not cause an increase in the number of leucocytes

in the blood. Tonsillitis *might* have caused a leucocytosis of that size, though it is generally smaller, but could not possibly have caused the tremendous drop in the red corpuscles. The operation was a cosmetic one, and the patient had been in sound health before it was done, so that his red corpuscles were presumably about 5,000,000. The only things practically which could have caused a fall from five to two millions in a few days were hæmorrhage—which had not occurred—and septicæmia. The interpretation was amply justified by the progress of the case.

Another case of a similar character was that of a lady, who had been confined about five weeks before I saw her. It was found later that the house was in a very insanitary state. She went on well at first, but about ten days after the confinement the temperature was found to be raised. She developed phlegmasia, first in one leg then in the other, but a gynæcologist who saw her found no local mischief in the uterus or pelvis, and the temperature began to fall. She got very pale, however, and exhibited the usual symptoms of anæmia. Her doctor thought she might be developing pernicious anæmia, and asked me to examine the blood. I found the reds to number 2,640,000, with 15 per cent. hæmoglobin, and a certain amount of poikilocytosis; the leucocytes were 13,400, and mostly polymorphonuclear, the fibrin was increased. Pernicious anæmia was therefore out of the question—the reds were too numerous, the hæmoglobin too low, so that the colour index was only 0·3, instead of being at least 1·0; while the leucocytosis, though it was not very high, and the increase of fibrin pointed to an inflammatory condition. I therefore reported that the case was a septic one, though the apparent absence of local mischief was puzzling, and treatment was carried on on that line. The patient lived for three weeks, during which I saw her several times, and in a second blood examination found the reds lower and the whites higher. *Post-mortem*, we found that the uterus was not enlarged, but that on its posterior wall were two small ulcers, and starting from them a thrombosis, with purulent softening, of the right uterine vein. This passed into the iliac vein, down both femoral veins, up the vena cava, and into the pulmonary artery.

The converse of these cases was found in a man who had a double pleurisy with effusion, with a febrile but irregular

temperature, a slight pericarditis, and an enlarged spleen. There was a vague history of rheumatism, but nothing else to point to a cause for the condition. Repeated blood examinations showed red corpuscles varying between 2,500,000 and 3,500,000, while the leucocytes were always below 5000. This last fact excluded rheumatism, where there is always a leucocytosis, depending in amount on the severity of the case, and excluded any septic element, while it made tuberculosis very probable. The man lived for two months, and then numerous miliary tubercles were found throughout the lungs and spleen, and a few in other organs. There is another point of interest in the case, and that is that this man had inflammation of three serous cavities, both pleuræ and the pericardium, and, in spite of this, there was no leucocytosis. Had the inflammation been rheumatic or septic, affection of a single cavity would have sufficed to produce a marked increase of the leucocytes.

A companion to this case is one which I saw a few days ago, and which also illustrates some of the difficulties of blood diagnosis. A little girl, aged ten, was taken ill rather suddenly about a fortnight before I saw her, and her temperature had remained high (about 103°) all that time. She had some abdominal tenderness, slight diarrhœa, with dark motions, some bronchitis, and lay in a semi-comatose state. The spleen was not enlarged, and there were no rose spots. The blood was repeatedly tested for the typhoid reaction, but the result had been negative. When I examined her blood I found the red cells little altered and numbering 4,080,000, while the white cells were 9400. This of course excluded any suppuration or septic mischief or pneumonia, but left undecided the question whether the disease was typhoid or general tuberculosis. I therefore made a careful differential count of the leucocytes in the stained films, and found that the polymorphonuclears numbered 80 per cent., the lymphocytes 20 per cent. Now the number of leucocytes, 9400, was not high for a child, and I did not think it could be called a leucocytosis. But the normal percentage of lymphocytes in a child of ten should be about forty instead of twenty as in this case, and in typhoid, apart from complications, the tendency is for the lymphocytes to increase in number. It may also do so in tuberculosis, but much less frequently. On the whole, therefore, as the Widal reaction was negative, and as enlargement of the spleen and

rose spots were both absent, I inclined to think the case one of general tuberculosis, and said so, but kept back in my mind the idea that there might be a commencing tubercular meningitis, as that is the one pure tubercular process in which a slight leucocytosis occurs. Two days after my examination the blood gave the Widal reaction strongly, and the case is now progressing favourably as a typhoid. The explanation of the relative leucocytosis, *i.e.* the increased percentage of polymorphonuclears, probably was that the bronchitis was more severe than one realised, and was producing this effect on the blood.

Another case illustrating the importance of leucocytosis was that of a girl, aged twenty-five, who was anæmic, slightly feverish, and had a markedly enlarged spleen and enlarged cervical glands. Her heart was enlarged, and there were slight systolic and diastolic murmurs. So far the condition might have been leucocythæmia, with a coincident cardiac lesion, but the blood was found to contain 18,000 leucocytes per cubic millimetre, and the vast majority of these were polymorphonuclears. There were no myelocytes, or nucleated red corpuscles, and no increase in the eosinophiles, as there would have been in a spleno-myelogenous leucocythæmia; and I hazarded the suggestion that the case was an endocarditis, which turned out to be correct.

I remember about six years ago passing two or three very unhappy days, which I might have avoided by a single blood examination. I was attending a family of children among whom measles had broken out. Three of them were attacked, and on the following day the eldest boy, whom I had not seen, so that he might have been feverish or not for all I knew, was taken ill with shiverings, cough, and high fever. At first I thought it was measles, as he had not had the disease, but the next day he was evidently very ill; the breathing had quickened, there was no rash, and no physical signs in the chest. Was it a severe aberrant form of measles, or a lobar pneumonia? The uncertainty lasted till the morning of the fourth day, when the familiar pneumonic signs showed themselves on one side, and the case went on satisfactorily. Had I counted the leucocytes on the first day I should probably have been able to make the diagnosis, for in measles there is no increase in their numbers; while in pneumonia, in 99 cases out

of 100, there is a well-marked leucocytosis from the very first. There is probably, therefore, no disease where the blood-count will more constantly give information than in central pneumonia without physical signs.

But leucocytosis is just as important to the surgeon as to the physician. If surgeons would condescend to examine the blood systematically, they would find that, for instance, in appendicitis, a leucocytosis increasing day by day would sometimes give them warning of pus-formation before the temperature or physical examination gave any sign of it, while all other septic conditions can be diagnosed from the blood more certainly than in any other way. The amount of leucocytosis present in malignant disease would give them an idea of the rate of growth and nature of the tumour, and of the existence or non-existence of metastases, and after its removal the reappearance of leucocytosis would warn them that the disease was recurring. In very mild and in very severe cases of pneumonia or other leucocytosis-producing diseases the blood gives a prognosis also. In obviously mild cases, where the patient easily overcomes the infection, the leucocytes are not increased, while in severe cases, where the infection easily overcomes the patient, there is no reaction and no leucocytosis. Such cases are doomed. In the great middle class, where there is leucocytosis, the actual amount of it depends on so many causes that it is impossible in most cases to draw a prognostic indication from it.

I might multiply cases which show the importance of leucocytosis indefinitely, but those I have narrated must suffice at present.

There are two diseases which are the very dust-bins of diagnosis, influenza in this country and malaria in America. It is worth remembering that neither of them cause leucocytosis, and that when we find a leucocytosis we cannot be dealing with uncomplicated influenza or malaria. I think all of us will be willing to confess that we are too ready to label anything that we are not sure of as influenza, and in another department of work I have scores of times seen febrile phthisis called by the other name, and have known all kinds of diseases, from appendicitis to tubercular meningitis, thrown into that dust-bin. In America the almost equal prevalence of malaria and typhoid leads to many mistakes, which might of course

always be cleared up by a blood examination, the plasmodium on the one hand, and the serum-reaction on the other, being sufficiently definite. In this country we do not very often see malaria, and we see even less of the plasmodium; for when a doctor finds that his patient has been in India recently, and has a febrile attack, his first instinct is to give him quinine, and it is not usually until a day or two afterwards that it occurs to him that it would be a good thing to have the blood examined. By that time, of course, the quinine has cleared the plasmodium out, if it ever was there. If you want to make a diagnosis of malaria, examine the blood, fresh or in films, *before* treating the patient.

The varieties of leucocytes present in the blood may give us useful information, and this must of course be sought in stained films. In passing, I may say to men who have no time for complete blood examinations, that they can learn more from stained films than from any one other method, and that if to that they add the enumeration of leucocytes, it will often be unnecessary to count red corpuscles, to estimate hæmoglobin, or even to examine a fresh film. With practice one can make a fair guess at the amount of hæmoglobin in the blood as it flows, and if one finds the red corpuscles normal in shape in the films, they are not as a rule greatly diminished in number; whilst by spreading films always as nearly as possible of the same thickness, one gets to know how near to one another the corpuscles should lie, and one can at once detect any considerable alteration in the number of leucocytes. The percentage of the different varieties of leucocytes can only be ascertained in films, for it is thoroughly misleading to attempt to estimate them from the number counted in the leucocytometer, though it is often stated in text-books that this is sufficient. One rarely counts more than a hundred leucocytes in that instrument, often not so many, and it is necessary to count at least thrice as many, and for real accuracy five hundred or a thousand. Further, in the leucocytometer one has not the staining reactions to guide one, so that a myelocyte looks just like a large lymphocyte, and an eosinophile like a "neutrophile." It is to be remembered, moreover, that the combination of eosin and hæmatoxylin is not a sufficient stain for diagnostic purposes; some combination of basic and acid anilin stains such as the triacid stain or eosin and methylene blue, must be used.

The following cases will illustrate the information to be obtained from stained films. About a year ago I saw a child with a large abdominal tumour on the left side. The doctor in charge of the case believed that this tumour was the spleen, and in shape and relations it was certainly very like it. He had examined the blood, had found the red corpuscles about 3,000,000, and the leucocytes between 30,000 and 40,000, if I remember aright. He had examined some films also, and thought that from them he got evidence that the disease was leucocythæmia. I saw the case, and as I knew his enumerations were likely to be accurate, did not repeat them, and merely made some films. When I examined these I found that there were no myelocytes and no eosinophiles, but that the majority of the leucocytes were polymorphonuclear neutrophiles, and the rest lymphocytes. It was therefore a leucocytosis, and not a leucocythæmia. I gave the opinion that the tumour was a sarcoma, probably of the kidney. At the *post-mortem* it was found to be a sarcoma of the suprarenal capsule, an extremely rare condition.

Another case which I did not see, and where the diagnosis, so far as I was concerned, was made from the films alone, was that of a gentleman who had been out of health for some time, but had not consulted his doctor. He became suddenly very ill, and his doctor found that his spleen was much enlarged and that his temperature was 105°. He sent for my friend, who afterwards gave me the films, but the patient was moribund, and died soon after. The films gave evidence of some anæmia; there were a few nucleated red corpuscles present, and in the three coverglasses which I received *every* leucocyte was a lymphocyte, and they were evidently increased in number. The explanation I gave was that the case was one of lymphatic leucocythæmia, in which the spleen is enlarged, the percentage of lymphocytes in the blood is often 98 or 99, and there is always some anæmia and a few nucleated red corpuscles; that the man had been attacked by some severe organismal infection, which had called out of his blood the few polymorphonuclear cells which were there, and that he had died before he had time or opportunity to manufacture a leucocytosis.

A very interesting series of observations is beginning to be made with regard to the eosinophiles. It has long been known

that they are increased in the blood and sputum in asthma, and that one can by that means distinguish true asthma from cardiac and renal dyspnœa. They are also increased in some skin diseases, notably in pemphigus and in dermatitis herpetiformis. In one case of the latter disease I found them to form 16 per cent. of the leucocytes present (the normal percentage is under 5), and have been able to show that two cases, supposed to be of that disease, were not so, by finding the percentages to be 4 and 5 respectively. But the disease which gives the highest percentage of eosinophiles is trichinosis—for some unexplained reason—and there they have been found in one case to make up 68 per cent. of all the leucocytes present. At first sight nothing seems more unlikely than that one should be able to diagnose such a disease by a peculiarity in the leucocytes, but the observation is quite recent, and it is an evidence that there is still much to be found in the blood if it be carefully and systematically examined.

The cases which I have chosen to illustrate my remarks have been selected in order to show that there is no branch of our profession which can afford to neglect the information to be got in this way. I do not mean, of course, that every patient who has a whitlow or a prolapsus uteri should have their blood examined, nor that any other means of diagnosis should be neglected, but I do plead for a systematic examination in all serious cases where there is any doubt about the diagnosis. The blood will often give positive information, and in many other cases will give negative information, which is often just as valuable. Those of the members of the Society who are familiar with the subject will of course realise that I have merely touched the fringe of it. My desire has not been to make an exhaustive exposition, but to stimulate interest and encourage observation.

Meeting II.—December 6, 1899

MR A. GORDON MILLER, *President, in the Chair*

I. ELECTION OF MEMBERS

Harold S. Ballantyne, M.B., C.M., B.Sc., Dalkeith; Alexander Hill Buchan, M.A., M.B., C.M., 42 Heriot Row; Charles C. Easterbrooke, M.B., C.M., Craig House, Morningside; David Huskie, M.A., M.B., C.M., Moffat; Hamilton G. Langwell, M.D., F.R.C.P.Ed., Leith; and John M'Gibbon, M.B., Ch.B., 55 Queen Street, were elected Ordinary Members of the Society.

II. EXHIBITION OF PATIENTS

1. *Mr David Wallace* exhibited a patient from whom he had removed a PYLORIC TUMOUR. Miss P., æt. 44, dated her symptoms from February 1899, when she suffered from indigestion. This improved under treatment, but in March the symptoms recurred and were more severe. No solid food could be retained in the stomach and she rapidly became weak and thin and markedly anæmic. A tumour was detected by palpation in the epigastric region which was very mobile and painless. Removal by Kocher's method was carried out by Mr Wallace on 15th June. The duodenum was sutured to the stomach as Kocher recommends. For some hours after operation she was very sick and vomited, but when turned on her right side this passed off and she made an uninterrupted recovery. Now, six months from date of operation she is in excellent health and can take ordinary diet.

2. *Mr H. J. Stiles* exhibited—

(*a*) A child, æt. 3 years, after operation for SEVERE DEFORMITY OF THE FOOT, following a burn, which occurred when the child was a year old. On admission to hospital it was found that there was a cicatrix extending from just below the knee, down the inner side of the leg, to the inner aspect of the ankle, where the cicatrix was dense and firmly adherent to the subjacent bones. The cicatricial contraction had caused great inversion of the foot, so that the sole looked inwards and a little upwards. The internal malleolus could not be made

out, and there was a history of several pieces of bone having come away from the inner side of the ankle. The heel was a little drawn up, and, as the child had never been able to bear any weight on the foot, there was no thickening of the skin or callosity on the outer aspect of the dorsum of the foot such as one sees in cases of congenital talipes-equino varus. The external malleolus and the lower part of the shaft of the fibula were very prominent, and arched outwards.

On September 28th Mr Stiles made a transverse incision across the inner ankle, and, with the aid of a periosteum elevator, separated freely the cicatrix for some distance from the lower part of the tibia. Next a longitudinal incision was made over the fibula, and an inch of the bone, just above the malleolus, was resected. The inner part of the wound was then retracted and the extensors and periosteum separated from the tibia, from the lower end of which a wedge was removed, the base of the wedge being directed outwards. The tibia was broken completely across at the level of the wedge. By further division of the soft parts at the inner ankle, the foot could now be forced outwards and the inversion corrected. When this was done it was found that the upper end of the lower fragment of the fibula became directed inwards away from the upper fragment; it was necessary, therefore, to wire the ends of the fibula. With the foot in good position, the skin over the external malleolus was redundant. A flap was shaped from the redundant skin, twisted round at its neck, and sutured to the edges of the gap at the inner ankle. The foot was put up in good position by means of an aluminium splint. Healing was uninterrupted except that the distal portion of the flap sloughed. Although the foot can now be placed with the sole flat upon the ground, the child will nevertheless be obliged to wear some form of ankle support to take the place of the structures destroyed at the inner ankle. The fibula is now firmly united.

Mr Stiles said that he had brought the case before the Society at this premature stage, because the parents, who are in very poor circumstances, came from the south of Scotland, and it was doubtful if the child would be able to make a second journey to Edinburgh.

In treating such joint deformities resulting from burns, Mr Stiles remarked that the surgeon should take advantage of

the great reparative power of the tissues in children. Amputation should be resorted to only after some such conservative treatment as above described had failed.

(*b*) Child with VENTRAL HERNIA after operation for suppurative appendicitis and general peritonitis.

The patient, aged 10 years, was admitted to the Children's Hospital on April 14th. She complained of severe pain in the lower part of the abdomen. She had walked all the way from Stockbridge in a "doubled-up" attitude. The history pointed to some acute abdominal mischief. Four days before admission she felt quite well, and indulged freely in skipping; the same evening she complained of pain in the abdomen. Next day she went to school although feeling very ill; she could take no food and began to vomit. Next day the pain became more severe, and the vomiting continued; the bowels had not been moved for three days. The following evening, that is to say, the evening prior to admission to hospital, she took a dose of castor oil which operated freely.

The patient walked into the out-patient room suffering severe pain, and looking exceedingly ill; face flushed; eyes sunken; temperature 100·6°; pulse 130; tongue furred. Further examination showed that she was unable to straighten her right leg; the abdomen was somewhat distended, with rigidity over the lower segment, and acute tenderness over the right iliac fossa. No induration or abscess could be felt. At 4 P.M. on the same afternoon the temperature was 101° and the pulse 144; the abdomen was fuller and more tympanitic. Having communicated with the parents, and obtained their permission for operation, the abdomen was opened in the right iliac fossa. A considerable quantity of sero-purulent fluid escaped, and the small intestines were here and there covered by small patches of purulent lymph. There was no pus in the iliac fossa; but on passing the finger into the adjacent part of the true pelvis there welled up a few ounces of thin, dirty-looking, and intensely fœtid pus. The appendix was not visible, neither could it be felt. The abscess, and the abdominal cavity, were washed out with large quantities of sterile water; two drainage tubes were introduced, one down into the pelvis and the other into the deepest part of the lumbar region. The rest of the wound was brought together with silkworm gut sutures. The child had a hard struggle for life; but the pulse

gradually improved, and, with it, the general condition. On the fourth day after the operation the suppurative process had spread in and around the wound, so that it was necessary to cut the stitches. The following day (the fifth), the child's general condition had so far improved that she was able to cry vigorously while being dressed, with the result that a coil of small intestine became partly prolapsed, necessitating the application of an iodoform compress dressing. During the next few days the patient's condition continued critical; the wound looked very unhealthy, and the suppurative process had extended into the neighbouring tissues of the abdominal wall; gradually, however, the local condition improved, and, with it, the general condition also.

On the 26th, that is to say, twelve days after the operation, the wound was beginning to contract; its surface, along with that of the protruding bowel, was covered with healthy granulations, and the sinus leading down into the pelvis had almost ceased to discharge. Improvement continued until May 9th (about a month after the operation), when the temperature went up to 101·4°. Next day the patient complained of abdominal pain; on the day following, vomiting set in, the pain became worse, the expression abdominal, and the breathing thoracic; pulse, 120. On the following day the patient was worse and the abdomen became more distended; pulse, 140; temperature, 102·6°. No flatus had passed during the previous twenty-four hours. At midday the patient was chloroformed in bed, and the finger was passed down into the pelvis through the wound, but no retained pus or other sign of fresh inflammatory mischief could be discovered. Mr Stiles saw the patient again early in the evening, and finding the symptoms still more marked and that a ladder pattern was distinctly visible, he opened the abdomen at the outer edge of the left rectus, expecting to find general peritonitis with possibly also some obstruction from adhesions. A quantity of clear fluid escaped; the small intestine was a little distended, but otherwise normal. The mesenteric glands were enlarged and evidently tubercular. Nothing to account for the patient's condition was detected, either in the region of the original operation, or down in the pelvis. The abdomen was therefore closed. From this date onwards the patient gradually improved and made a slow but uninterrupted recovery. The

original wound gradually cicatrised over. The patient left hospital three months after admission, with a ventral hernia.

Three months later (October 9th) she was again admitted into hospital in a very critical condition, this time with a left-sided acute empyema. She was cyanosed, very breathless, and her pulse was very shabby. On examination it was found that there was a lax, partly reducible, fluctuating swelling over the second left intercostal space, anteriorly. The physical signs indicated that the left pleural cavity was full of fluid. Twenty-two and a half ounces of pus were drawn off by aspiration. The following day a portion of the eighth rib was resected, and a drain introduced. She made a good recovery, and was discharged five weeks after admission looking fat and well.

Mr Stiles remarked that he thought the knife might claim to have rescued the patient on no less than three occasions within a year. It was to be hoped that she would be spared its use for all time to come.

(*c*) Infant after operation for ACUTE INTUSSUSCEPTION, which was, apparently, secondary to acute appendicitis.

The infant, aged 3 months, strong and well-nourished, was admitted to the Children's Hospital at 1 P.M. on September 28th. At 2 P.M. the previous day the patient was seized with violent spasmodic pain in the abdomen; at 4 A.M. blood-stained mucus began to pass by the rectum. Dr Findlay, who saw the patient in the forenoon, felt a swelling in the right iliac fossa, and, suspecting intussusception, sent the child at once to hospital.

On admission the patient looked distressed, but not seriously ill. The abdomen was quite natural in appearance, but too rigid and tender to admit of thorough palpation. An anæsthetic was given, and permission obtained to proceed at once with operation if deemed necessary. On palpation, the bowel, corresponding in position to the cæcum and ascending colon, appeared to be more distinctly felt than natural, as if thickened, but it did not give the feel of a firm sausage-shaped tumour, such as one so frequently meets with in intussusception. The rectum contained a quantity of blood-stained mucus, but no invaginated bowel. After some little hesitation, Mr Stiles decided to open the abdomen in the middle line immediately above the umbilicus. Two or three ounces of yellowish

transparent fluid escaped. The small intestine was a little congested, otherwise normal. Exploration with the finger detected no intussusception, but the cæcum and lower end of the ileum felt decidedly thicker and firmer than normal. The wound was enlarged, and, after pulling out part of the small intestine, the cæcum and lower end of the ileum were brought into view; the whole of the former, the lower 4 inches or so of the latter, and the commencement of the ascending colon were, as has been already stated, thickened, firmly œdematous, and covered here and there with recent lymph. The ileo-cæcal junction was markedly dimpled towards the cavity of the cæcum. On examining the appendix, the distal half was found to be much swollen and intensely conjested; the proximal half was normal, and there were no adhesions or thickening of the mesentery of the appendix, or any distension of its veins.

The appendix was amputated in the usual manner, and, after suturing a small tear in the peritoneum of the cæcum, the abdomen was closed. A few hours after the operation the infant took the breast heartily. The little patient made a complete and uninterrupted recovery, and left the hospital on the twelfth day.

Although the physical signs were not sufficient to make the diagnosis of intussusception certain, Mr Stiles deemed it right to make a diagnostic incision, as he felt that, should the infant be suffering from intussusception, early operation gave the only chance.

Having regard to the symptoms, to the slightly invaginated appearance of the ileo-cæcal junction, and to the indurated and œdematous condition of the gut, Mr Stiles thought that there could be no doubt that the infant had suffered from an intussusception which had become reduced. The condition of the appendix was not that of simple venous congestion, secondary to the intussusception, but of an acute primary inflammation of its distal half. The probability, therefore, was that the appendicitis was the exciting cause of the intussusception. If this view be correct, Mr Stiles thought that the case was worthy of record, as an instance of acute appendicitis occurring in an infant who was being brought up on the breast; it is possible also that this patient, aged three months, is about the youngest who has undergone the operation of appendecectomy.

The case also well illustrates the gain to be derived from a diagnostic incision in obscure abdominal conditions, even in young infants. Lastly, Mr Stiles remarked that in infants and young children it is often better to make the mesial diagnostic incision above rather than below the umbilicus—(1) because the umbilicus at this age is placed relatively much lower than in adults; (2) because the whole abdomen, including the pelvis, can readily be explored. In this particular case no difficulty whatever was experienced in removing the appendix. In cases, however, where appendicitis is suspected it is, of course, advisable to make the incision in the right iliac fossa.

3. *Dr Scott Skirving* exhibited—

(*a*) A patient operated on by him in Leith Hospital in 1897 for RUPTURED APPENDIX with GENERAL PERITONITIS.

The patient, æt. 22, was an example of the fulminating type of appendicitis. The usual symptoms had been marked and severe and the onset sudden. On his admission on the third day he was unconscious and profoundly collapsed. His condition was such that no hope was entertained of his recovery. Operation, however, was performed at once. Very offensive pus was found and much recent lymph. The only region of the abdominal cavity not affected was the upper half of the left side. Nearly 2 pints of fluid were present, mostly in the pelvis. The appendix was ruptured about a ¼-inch from the cæcum and hung by a shred of necrosed tissue with the lumen exposed. No attempt was made to clean all the coils of intestine, but copious washings-out with very hot water were employed, and the patient vigorously stimulated by the usual methods, both during and after operation. The freest possible drainage was provided, and the large wound hardly stitched at all. The stump of appendix was ligatured and a fold of cæcal wall rapidly stitched over it, so as to cover it. Improvement was marked next morning and increased from day to day. Discharge from the wound was long continued. The patient had now been engaged in active work for more than a year. Much less tendency to hernia existed than might have been expected. The long recumbency might account for this.

(*b*) Patient after SUBPERIOSTEAL RESECTION of LOWER JAW (one half).

The operation was performed two months previously on a

girl, æt. 4, for necrosis following injury. The operation was completely subperiosteal, and the cavity of the mouth was not opened into. New bone had formed very early, and was now abundant. Chewing, etc., was done very satisfactorily. On the other hand, the lob-sided appearance of the face, frequently seen after excision, due to the projection of the jaw to the affected side, was marked. If this was due to the unopposed pterygoids on the healthy side, could it be prevented in any way at the time of operation, or by the application of some kind of splint afterwards? Improvement had been hoped for in this (subperiosteal) case, where the insertions of the pterygoids had not been divided. Some facial paralysis had appeared a day or so after operation, apparently inflammatory in origin. A slight amount still persisted. Possibly some twigs of the nerve had been caught in the new bone formation. A further point of interest was that a small amount of discharge (not saliva), still came from a corner of the cicatrix. Had the subperiosteal method, therefore, been overdone?

III. Exhibition of Pathological Specimens

1. *Mr David Wallace* exhibited—

(*a*) Tumour of pituitary body. W. T. was admitted into Ward XIII. of the Royal Infirmary on February 12th, 1899, in a comatose condition. He had been found at the foot of a stair down which he was supposed to have fallen, but as it was known he had been drinking he was put to bed on the supposition that he was intoxicated. Twelve hours later he was brought to the infirmary. On admission: temperature, 97°; pulse, 70; respirations, 30, very stertorous, and accompanied by flapping of cheeks. Pupils equal and moderately contracted with no reaction to light. Reflexes exaggerated. Limbs completely rigid. There was no sign of injury to the head, no bleeding from the ears. The condition was thought to be most probably a laceration of the brain with surface or ventricular hæmorrhage, and as he seemed *in extremis*, no operation was advised. No change occurred in the symptoms, and he died seventy-two hours from the time of injury. The temperature rose to 105·2° before death. At the autopsy a tumour the size of a tangerine orange, replacing the pituitary

body, was found and no other brain lesion. The sella turcica was markedly enlarged.

(*b*) CEREBELLUM WITH CALCAREOUS CONCRETION, size of a marble. J. W., æt. 18, admitted 16th July 1899, had double otorrhœa of long standing (three years at least). Ten days before admission she felt a sudden severe pain in the left ear, which still continues, although rather less painful. On 18th July, temperature, 99°; pulse, 102; respirations, 24. During stay in hospital been vomiting occasionally, quite apart from meals. No œdema behind ear on either side, but still complains of great pain on left side of head. Operation on this day. The left middle ear had granulations and fœtid pus. Mastoid Antrum and lateral sinus apparently healthy. For a day or two she improved, but on 23rd temperature rose to 103·6°; pulse, 88 to 104; respiration, 32 to 42; and as her condition seemed getting progressively worse the right ear was operated upon. The middle ear contained pus and granulation tissue, but the antrum and lateral sinus seemed healthy. She gradually got worse, but as no optic neuritis was present, and as there were no localising symptoms no further operative treatment was resorted to. She died on the 24th July.

Autopsy.—No leptomeningitis nor pus in brain. Lateral sinuses healthy. Indeed, no cause of death discoverable, but in the left cerebellar lobe, which was markedly atrophied, the calcareous concretion shown was found embedded.

(*c*) FRACTURE OF SKULL almost completely across ANTERIOR FOSSÆ implicating left middle meningeal artery. J. R., æt. 14, admitted 16th September 1899, had fallen a distance of 20 feet, and struck his head over frontal region about one hour before. He was unconscious, with pale face and sighing respirations. Pulse 110 and weak. Pupils, equal and reacting to light (*concussion*). Vomited shortly after admission, the ejected matter contained no blood. There was a curved wound 3 inches long over the left frontal eminence. The bone was bare but no fracture. No escape of cerebrospinal fluid from ears and no bleeding from nose. Recovered consciousness and complained of pain in the right arm, but could move all his limbs freely. This half an hour after admission. Mr Wallace expressed the opinion that there might be injury to one of the meningeal arteries, and that a careful watch for localising symptoms was to be kept. During the night the

right thumb twitched once or twice, but no other movements were observed. The pupils remained equal and moderately contracted, and some ecchymosis appeared under the right conjunctiva. He slept well and at 9 A.M. next morning, twenty-two hours from injury, was quite conscious and spoke to those around him. Temperature, 98·6°; pulse, 108; respirations, 24.

9.10 A.M. Vomited bile and milk.

9.15 A.M. Noisy and restless, tossing all his limbs about the bed and complaining of pain in his head. Pulse now 60. Pupils rather dilated, but equal and react to light.

10.30 A.M. Much worse, comatose, pulse 120, both arms jerking.

11 A.M. *Left* pupil widely dilated and fixed. *Right* contracted.

Mr Wallace saw the patient at this hour and decided to operate. From the pupil condition and the jerking of right thumb which had been observed during the night, the skull was opened over the left middle meningeal (ant. inf. angle of left parietal bone). On turning down a flap separation of the coronal suture observed, and when circle of bone removed a large extra dural clot was exposed. The clot was partially scooped, partially washed out. It was especially over the frontal lobe, which was markedly flattened. Only the most posterior part of the clot reached the Rolandic fissure, and that at its inferior end. After removal of the clot the brain gradually expanded. The left pupil contracted and the breathing became less stertorous. The lad died six hours later.

Autopsy.—Separation of coronal suture completely across the vertex, and a fissured fracture running through the orbital plates on each side were discovered. This fissure passed into the middle fossa on the left side so far as to implicate the middle meningeal artery. The brain was very slightly lacerated on the under surface of the *left* temporo sphenoidal lobe, and there was a slight laceration on the under surface of the left cerebellar lobe. At those parts, however, practically no hæmorrhage had occurred.

This case seems of special interest in demonstrating that great compression of the frontal lobe can occur without symptoms, and yet how suddenly symptoms of great gravity and depth can supervene when the clot reaches a certain size. The patient was carefully and specially observed with the idea that middle meningeal hæmorrhage might occur, but nothing occurred to indicate that it had.

(*d*) TUMOUR OF PYLORIC END OF STOMACH, from patient shown at meeting.

(*e*) IMPACTED CALCULUS IN URETER, from a patient who had hydronephrosis. A stone was removed from the pelvis of the kidney. The patient progressed favourably for three days when suppression of urine supervened and lasted for four days. On the eighth day after operation urine was secreted, but just when the patient seemed to be improving he died quite suddenly. At the autopsy the specimen shown was found in the ureter of the kidney which had been operated upon. The other kidney was healthy macroscopically as well as microscopically.

2. *Mr Alex. Miles* exhibited—

(*a*) A large CHONDRONA growing from the posterior aspect of the femur.

(*b*) The CÆCUM, ASCENDING COLON and PORTION OF THE TRANSVERSE COLON (measuring in all 23½ inches), removed from a man, aged 32, for malignant disease (carcinoma). The transverse colon and small intestine were united end to end by six stitches. The patient made good progress for six days and then suddenly began to sink and died on the eighth day. At the post-mortem no cause for death could be found.

(*c*) The CÆCUM, ASCENDING COLON and PORTION OF THE TRANSVERSE COLON (measuring 22 inches), removed from a man, aged 51, for carcinoma. End to end anastomosis was made in this case also with silk. The bowels moved satisfactorily on the third day; the patient was on the sofa on the sixteenth day, and has resumed his work as a miner.

(*d*) A series of specimens illustrating injuries to the skull, including (*a*) two extensive basal fractures; (*b*) a gun-shot wound of the skull; (*c*) an injury to the skull and brain produced by a large piece of steel; (*d*) two cases of middle meningeal hæmorrhage.

These specimens were illustrated by stereoscopic photographs.

3. *Dr Harvey Littlejohn* exhibited—

(*a*) A DERMOID CYST OF THE LEFT OVARY. The woman, æt. 39, died suddenly from heart disease. At the post-mortem a tumour the size of a large orange was found lying above the

cœcum. It was perfectly free and had a pedicle 5 inches long. The contents were fluid, and on opening the cyst it was found to contain fatty matter mixed with hairs. In the wall near the base were two teeth. There was also a fibro-myoma of the uterus, the size of a fœtal head. The right ovary was normal. There were no adhesions.

(*b*) Heart showing almost COMPLETE CALCIFICATION OF THE COLUMNÆ CARNEÆ OF THE LEFT VENTRICLE. The specimen was obtained from a woman—unmarried—æt. 30, who dropped down dead in the street. She had suffered five years ago from anæmia, but otherwise had been always in good health and continued in her employment of a domestic servant. A week previous to her death, her sister thought that she was again becoming anæmic and took her to see a doctor who carefully examined her but could make out nothing the matter with her heart except that the heart sounds were somewhat weak. There were no murmurs. Her mistress states that on the morning of her death she was apparently perfectly well, cheery and active. She had never suffered from rheumatism or any other febrile illness that her relations can recall.

At the post-mortem the heart was seen to be enlarged, the left ventricle being increased in size and presenting a somewhat abnormal outline. The right coronary artery was greatly dilated, sinuous, its walls thickened in patches, but not calcareous. On opening into the left ventricle, the walls were seen to be hypertrophied, the cavity dilated and rigidly distended by a mass of calcareous matter, which encircled the ventricle immediately below the level of the aortic orifice. The calcification was branching, having apparently taken place for the most part in the columnæ carneæ, and it completely prevented contraction of the upper half of the ventricle. The aortic valves were healthy. The mitral valves showed some recent vegetative thickening. The wall of the left auricle contained one or two patches of calcareous deposit. The aorta showed one or two atheromatous patches, but no calcareous deposit. The other cavities presented a healthy and normal appearance.

4. *Dr Scot Skirving* exhibited—

(*a*) NECROSED LOWER JAW of patient exhibited at same meeting.

(*b*) RUPTURED APPENDIX of patient exhibited at same meeting.

(*c*) RUPTURED APPENDIX of large size with typical concretion *in situ*. This case in nearly every respect formed a marked contrast to (*b*). The reason he had decided to operate was the presence of intense local tenderness at M'Burney's point. Even light percussion could not be borne. The other symptoms were absent or only slightly marked. Only a few drops of pus were present. The thickened appendix lay immediately under the parieties, fixed by recent adhesions, and, until the wound was enlarged, was thought to be the cæcum. The patient made a rapid recovery.

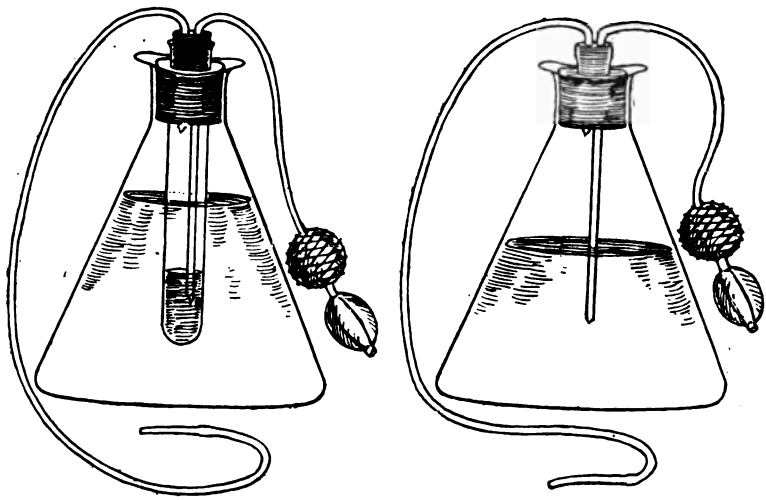
5. *Dr G. L. Gulland* exhibited a series of microscopic preparations made by Cohn's method for the demonstration of fat. The preparations were from chylous and chyliform ascites, from pleural effusion in a case of sarcoma of the lung, from urines containing tube casts, and the débris from the bladder in a case of villous cancer. Cohn's method appears in *Zeitschr. für Klin. Medicin*, 1899, and consists in fixation of dried films in a 10 per cent. watery solution of formalin, staining first in a concentrated solution of sudan iii in 70 per cent. alcohol, and then in hæmatoxylin, and mounting in Farrant's medium. Fat is stained orange-red, to which the blue colour given by the hæmatoxylin forms an excellent background.

IV. EXHIBITION OF INSTRUMENTS

Dr A. Douglas Webster exhibited a hot-air spray for volatile medicaments. This consists of an Erlanmeyer's flask of capacity about 500 c.c., into the neck of which is fitted a cork with a hole in the centre. Into this is inserted a test-tube closed by a rubber cork having two holes in it. Through these holes are fitted two finely-pointed glass tubes, one of which passes nearly to the bottom of the test-tube, while the other only just passes through. An ordinary rubber bellows is attached to the longer piece of glass tubing, and to the other is fastened about a foot and a half of rubber tube. The test-tube being removed, the flask is three-quarters filled with water of the desired temperature, or which may be heated by a spirit-

lamp. The volatile medicament, or one dissolved in any fixed oil, is then poured into the test-tube, filling it about a quarter full, so that the long glass tube passes below the surface of the fluid. (Solutions in spirit or water are naturally not suitable for a dry hot-air spray.) The test-tube is then replaced, and the medicament is soon warmed to the temperature of the water, which is usually from 120° to 160° F., the effect being that when the bellows are worked a very finely divided and warm spray of the pure remedy is driven out in the form of a cloud. Many different remedies which could be used in this apparatus were thus exhibited.

Among these was one composed of a mixture of turpentine



two parts, eucalyptus oil one part, and a 10 per cent. solution of menthol in parolein part which had been found useful in several cases in adults of chronic eustachian deafness, and in children in enlarged tonsils and adenoids with deafness, in both cases relieving and removing catarrh. Pure cresolene, cresol and other coal-tar products thus vapourised were found effective in whooping-cough and diphtheria. Menthol in parolein, or in olive, almond or vaseline oil 5 to 15 per cent. (the latter of which is liquefied by the heat of the water), as mediums, were specially satisfactory.

These as well as pure terebene, pumilene and pinol were found applicable in catarrhs. In ozena, septic sore throat and phthisis sprays of pure carbolic acid or creasote had been tried

with success. Turpentine, cajuput, pine, eucalyptus, gaultheria and other aromatic oils were also exhibited.

The spray is of simple construction, and by a slight adaptation of the cork the apparatus could also be used as an ordinary vapour spray and inhaler, which would appear to be particularly efficacious, as the volume of the vapour is under control, and can be directed to the affected area of mucous membrane. This adaptation also is suitable for the application of remedies dissolved in water of spirit.

Ipecac. wine in croup and compound tincture of benzoin in tonsillitis had been useful, one teaspoonful of either in the flask, one half filled with hot water at the temperature of about 140° F.

V. Original Communication

ON THE RADICAL CURE OF HERNIA, WITH THE STATISTICS OF SEVEN YEARS' EXPERIENCE IN THE ROYAL INFIRMARY

By Charles Watson MacGillivray, M.D., FR.C.S.Ed., Surgeon, Royal Infirmary, Edinburgh

So much has been written of late years regarding the radical cure of hernia, and so many new methods have been devised for its accomplishment, that one almost feels further writing on the subject to be superfluous.

But there is little doubt that in this, as in so many of the new operations which the improvements of modern surgery have introduced, more especially in the region of the abdomen, the tendency has been to give up the older and more expectant methods of treatment, and for all and sundry, without special knowledge or experience, to have recourse to what can only be regarded as rash and unnecessary frequency in operating, where milder measures would have led to safer and often more permanent results. In hernia the almost certain primary safety of the operation and the immediate temporary improvement which result from it, in the hands of the skilled operator, have led to the laying down of general rules of treatment, which may become unsatisfactory, and even dangerous, when applied by those without much surgical experience.

A clear understanding of the value of such modern opera-

tions can only be attained by extensive individual experience, which alone can teach what are the immediate dangers, and what the ultimate prospects as to permanent relief when they are employed.

Statistics and general rules can thus only be formulated from the results of individual experience; and it seems to me that those who happen to be placed in positions where frequent opportunities for seeing such cases and operating upon them occur are bound to add their observations to those of others. It is for this reason that I think it right to give here an account of the cases of this description which I have seen during the seven years I have been a hospital surgeon.

Before, however, taking up individual operations, it will be well to consider the subject in its general aspects.

First, then, as to the *class of cases and conditions of hernia* in which an operation for a radical cure is advisable.

Here one must first take into consideration the *general condition of the patient*, quite irrespective of the local state. It is hardly necessary to say that, unless in very exceptional circumstances, one would never think of operating where organic disease was present, or a very poor state of the general health.

As to *age*, youth is no hindrance to operation. Indeed, in those frequent cases where, in infants suffering from congenital hernia, social surroundings prevent the satisfactory application of a truss, excellent and safe results can be attained by operation. But in young adult life, when the health is good and the muscles strong, and where the hernia is of recent formation, is found the *beau ideal* case for operation. Those patients who are past middle life, and who have pendulous bellies and weak muscles, especially when they have wide, opened up canals and large, long-standing ruptures, should rarely be subjected to operation. The results are uncertain, and the dangers considerable.

The *sex* of the patient is of little importance, excepting in that it affects the form of hernia which is present.

We have only really to consider three forms. *Inguinal*, generally in the male, rarely in the female. *Femoral*, commonly in the female, not unfrequently in the male. *Umbilical*, practically confined to infants and middle-aged or old women, and seldom calling for operation, except in the case of the latter.

Taking, then, these general considerations into account,

along with the condition of the hernia, we may divide the cases which are suitable for the operation for radical cure into those in which operation comes to be either *imperative*, *necessary*, *advisable*, or *convenient*.

1. *Imperative.*—The cases of hernia in which operative treatment becomes *imperative* are those in which there is strangulation or some other complication, and in which, if no operation were performed, death would inevitably result. In such the question of *radical cure* is only secondary, and whether or not it is justifiable to attempt the operation for radical cure depends upon the condition of the parts and the state, age, and circumstances of the patient. In all these cases the operation should be as simple as possible.

2. *Necessary.*—Sometimes operation is *necessary*, although not imperative. Cases which come into this category are those in which the hernia cannot be kept up by means of a truss; this makes the condition of the patient one of constant danger, accompanied by pain and inconvenience, and commonly renders him unfit for his ordinary work.

3. *Advisable.*—Less urgent than either of the preceding groups are cases in which, although operation is by no means necessary, it may still be, and ought to be recommended. These are found, for the most part, amongst patients of the poorer class who have to work hard. They are cases in which a truss would be amply sufficient for a patient leading a sedentary life or a life of ease, but in which, during the severe exertion of body to which poorer patients are often exposed at their work, the hernia would not be sufficiently controlled for safety. With these may be included those congenital hernias in which the poverty of the patient's circumstances prevents the acquisition in the first place of a suitable truss, and in which, even if a proper truss were used, the constant care of it which is necessary to the production of a permanent cure could not be guaranteed.

4. *Convenience.*—Where a truss acts quite satisfactorily, but the patient does not care to put up with the inconvenience of any apparatus, and wishes to be permanently relieved, even at some slight risk, an operation of *convenience* may be performed. And in this group are cases also in which the presence of a hernia, however slight it may be, proves a bar to congenial occupation or to the public services.

We have next to consider the question of the *danger to life* of the operation. Here, as in appendicitis, and many other conditions, two great divisions must be made. First, there are cases where, as in strangulation, some operation is imperative, and where the question of radical cure is only a secondary consideration. Here the danger depends not so much upon the operation itself as upon the local and general condition of the patient, although the superadding of an operation for radical cure may add somewhat to the whole danger. And second, are the cases when the patient is in perfect health at the time of the operation. It is obvious that these two classes cannot be regarded as being upon the same footing.

The age of the patient naturally influences the danger and the mortality. Youth, even infancy, does not appear to add to the danger. The mortality of uncomplicated cases in young subjects is practically nil. To even a greater extent the same holds good in early adult life. After middle life the danger increases, and in old age it is considerable, so that the operation should not then be undertaken rashly, especially if the patient's general health be not good or if the hernia be large or complicated.

But, quite apart from the question of immediate danger, the *ultimate result and its permanency* require consideration, and here quite a number of points arise as influencing the prognosis. Of these the most important are the age of the patient and the method employed, the nature of the rupture and of the surrounding parts, the mode of healing and the after-treatment.

Age has already been discussed as a factor of importance, but it may be added that, so far as the ultimate result of the operation is concerned, the earlier the patient is operated upon the better, because, with the growth of the patient there is a natural tendency to cure. In fact, in many cases of congenital hernia, operation is unnecessary, a radical cure usually following the careful application for a year or two of a well-fitting truss. In such cases the complete obliteration of the open funicular process does not necessarily follow, and successful operation would secure the most certain good result, yet one is not justified in urging it except in those cases in which the state of the hernia or the social surroundings of the

little patient, negatives the probability of a cure by palliative measures.

In considering the *method to be employed*, so long as the primarily important point of the obliteration of the sac is carried out, the rest is very much a matter to be decided by individual experience and taste.

One may say, generally, that in children and imperative cases, the simpler the operation can be made the better. In young and healthy individuals more thorough and more complicated methods may be employed. In cases of large, old-standing hernia, with dilated canals, if an operation be undertaken at all it should be a very complete one, if a permanently good result is to be looked for.

The treatment of the sac which I usually employ consists in clearing it from the constituents of the cord as far up as the level of the internal ring, transfixing, and tying it with chromic catgut. The long ends of the catgut are then passed, by means of M'Ewen's needles, the one end through the lower edges of the transversalis and internal oblique, and the aponeurosis of the external oblique, about an inch to the outer side of the internal ring ; the other, at the same level, through Poupart's ligament. When the two ends are brought together and tied, they not only draw the stump of the neck of the sac up towards the anterior superior spine of the ilium, but also strengthen the outer half of the circumference of the internal ring. This part of the operation is easy, except in the case of congenital herniæ, where the separation of the open funicular process from the spread out constituents of the cord is at times difficult, and, naturally, requires a division of the sac low down, so as to leave a tunica vaginalis, the upper end of which is sown up with fine catgut.

As to the closure of the canal, in infants and in cases of strangulation, I do not recommend the splitting up of the anterior wall. In the former, a few sutures of chromic catgut, passed through the aponeurosis of the external oblique, catching up the lower edges of the internal oblique and transversalis, and passing in front of the cord—protected by the finger—out through Poupart's ligament, does all that is necessary. In the latter the same method may be employed, but in severe cases and in old people the canal had better be left alone to contract of itself.

In young healthy individuals, or where the canal is large, a modification of Bassini's method may usefully be employed. The anterior wall is slit up to nearly the level of the internal ring, the cord is lifted from its bed, and the lower edge of the internal oblique and transversalis, with their continuations as the conjoint tendon, are sutured, with interrupted sutures of chromic gut to Poupart's ligament, right up to the level of the internal ring. The cord is then replaced, and the lower edge of the upper border of the divided aponeurosis of the external oblique is also sutured to Poupart's ligament, whilst the upper edge of the lower border of the aponeurosis is sutured to the anterior surface of the aponeurosis, after the fashion of a double-breasted waistcoat.

In the case of a femoral hernia, the neck of the sac, having been cleared as far up as possible, is transfixed and ligatured with chromic gut. The two long ends of the ligature are then, by means of M'Ewen's needles, passed through the anterior wall of the abdomen, just above the inguinal canal, about an inch apart, and are tied together, so as to displace the neck of the sac from its position as regards the crural canal. This seems to be the essential part of the operation, and I have never seen any tendency to relapse. The closure of the canal is notoriously difficult. One or two chromic gut sutures are passed through the fascia over the pectineus and Poupart's ligament, so as to close the greater part of the canal at the inner side. Sutures cannot be safely introduced towards the outer side on account of the proximity of the femoral vein exposing it to risk of injury.

This method I have found less troublesome and quite as satisfactory as that of Watson Cheyne.

In umbilical hernia the method to be employed must be modified to suit individual cases; but, speaking generally, it consists in opening the sac, removing superfluous omentum, relieving adhesions, and replacing the contents. In so doing an elliptical portion of skin has generally to be removed. When possible the neck of the sac is ligatured, or else the edges of the opening in the peritoneum are brought together by means of catgut sutures. The edges of the two recti, when not too far apart or atrophied, are to be rawed and united by chromic gut. Finally, the skin wound is stitched up.

In the matter of the *mode of healing* I cannot say that I

agree with M'Burney that healing by granulation is advantageous, and that the firm mass of cicatrix thus produced prevents relapse. The only two, out of all my cases, that have returned complaining of a relapse were those that, from some cause or other, became septic, most unfortunately, and suppurated. These cases took long to heal, and there was a large amount of cicatricial tissue formed. But this tissue had subsequently stretched and bulged, giving rise to uneasiness and protrusion. I believe, then, that healing by first intention is of the utmost importance and that failure in this respect is one of the most frequent causes of subsequent relapse.

A point of great importance in the *after-treatment* is keeping the patient in bed, or, at anyrate, in the horizontal position, for six weeks after the operation. Till that time has elapsed the wound, although united, is not firm and is therefore liable to stretch.

When everything has gone well, I do not think that the use of a truss is advisable. The continued pressure of it is apt, through producing pressure atrophy, to weaken the resisting power of the cicatrix. When violent efforts have occasionally to be made, a light truss may be worn for the time being, so as to give a passing support.

The question which is most difficult of all to decide by reliable observation is the question of permanency of results. Patients upon whom an operation for radical cure has been performed are like patients with stricture; however strongly one may impress upon them the necessity of reporting themselves from time to time, they practically never come back, in my experience, unless something goes wrong. I have happened to see several of them months and even years after the operation, and have found no relapse; yet the only ones who have officially returned to report themselves are the two in whom abscesses had formed, and in whom the ultimate result was unsatisfactory. One cannot, of course, hope that in the case of all the others their non-return indicated permanent cure, but it is worthy of note that the only two who did return for further advice were the two about whom one feared failure.

Now we pass to the real object of this paper, namely, to detail the statistics of the operations for radical cure which have been performed in my wards during the past seven years.

In all there have been ninety-one operations, with ten deaths.

Of these deaths eight occurred in cases operated upon for strangulated hernia, in which the symptoms were severe and the prognosis bad before operation, and in which the question of the radical cure had nothing to do with the unfortunate result. Two deaths occurred after the operation for radical cure, uncomplicated by strangulation. The first of these was in the case of a strong young man, in whom persistent vomiting set in, as he was recovering from the effects of the anæsthetic, and continued until he died, some days later. There was no abdominal pain nor symptoms of internal obstruction, nor was there any peritonitis, and the wound healed by first intention. The other case was that of an infant, aged 3 years, on whom a double operation for large congenital inguinal herniæ was performed, and who died ten days later of double pneumonia, the wounds in this case also having healed by first intention without a bad symptom.

Of these ninety-one patients seventy suffered from inguinal hernia, seventeen from femoral hernia, and four from umbilical or ventral hernia.

Inguinal Hernia.—Seventy cases with six deaths; four of the fatal cases being complicated by strangulation, all four being males, aged 64, 28, (?), and 13 respectively. Fifty cases were simply operated upon for radical cure. Fourteen were complicated by strangulation, five by irreducible hernia, one by incarceration. Twenty cases were congenital, four of these being in young male adults. Seven cases were double, two in male infants; two in adult females, aged 21 and 36; three in adult males, aged 38, 23, and 21. Three cases were in females.

As regards age—nineteen cases were under 20 years of age; twenty-eight were between 20 and 40; nine between 40 and 60; eight between 60 and 80. In six the age is not stated.

Femoral Hernia.—Seventeen cases with three deaths, all females, aged 34, 64, and 66, and all cases of strangulation. Eight cases were simply operated upon for radical cure. Eight were complicated by strangulation; one by incarceration. Six were males, in two of whom the hernia was strangulated; eleven were females, with six cases of strangulation and one of incarceration.

As to age—none were under 20. Between 20 and 40

there were three females and one male; between 40 and 60 six females and five males; between 60 and 80 two females.

There were no cases of double femoral hernia.

Umbilical and Ventral Hernia.—Four cases, all in fat, middle-aged married women who had borne children. Three recovered and one died. Two, aged 45 and 64, were operated upon for the radical cure of irreducible ventral herniæ. One, aged 45, was operated upon for irreducible ventral hernia which had become strangulated. One, aged 48, who was operated upon for an incarcerated and inflamed hernia, died.

The three who recovered were provided with belts, and in none of them was there any recurrence some months after the operation.

These results would show a death rate of a little over 3 per cent. in all the cases, favourable and unfavourable, which were operated upon for radical cure simply, the exact numbers being two deaths in sixty cases.

In the thirty-one cases which were complicated by strangulation or a similar severe condition the total number of deaths was eight, or about 25 per cent. In none of these could the simple means employed for the radical cure be blamed for the fatal result, because the general or local condition of the patient before the operation was, in each case, amply sufficient to account for the death.

There is one really unsatisfactory feature of these statistics, and that is the absence of information as to the ultimate results of the operations so far as permanent cure is concerned. But I hope to be able at some later period, having followed up those of the cases which are accessible, to embody in a short supplementary report an account of the ultimate results.

In any case my experience seems to prove that, in suitable cases, the danger of the operation is slight; while the immediate results, so far as regards the comfort and well-being of the patient, are excellent.

Meeting III.—December 20th, 1899

MR A. G. MILLER, *President, in the Chair*

I. EXHIBITION OF PATIENTS

1. *Dr Alexander Bruce* exhibited (*a*) a patient, aged 17, with INFANTILE PARALYSIS limited to the intrinsic muscles of the left hand. All the muscles of the thenar and hypothenar eminences, as well as the interossei and lumbricals, were completely atrophied, and failed to give the slightest response to either galvanic or faradic stimulation. The muscles of the left forearm were somewhat smaller in size than those of the right, but the reduction was not greater than could be accounted for by the lessened use of the hand, and there was no alteration of the electrical reactions. There was no sensory loss in the hand, showing that the condition could not have resulted from a peripheral neuritis of the ulnar and median nerves. The condition had appeared during the first year of life, and had been attributed to an injury to the hand—which was manifestly not accountable for it.

(*b*) A man showing typical symptoms of CONVULSIVE TREMOR or MYOCLONUS MULTIPLEX, which will be described *in extenso* in the *Scottish Medical and Surgical Journal.*

(*c*) A man with signs of INJURY TO THE LAST DORSAL, all the lumbar and the first sacral nerve roots on the side, causing loss of power in the left leg and anæsthesia corresponding to the affected roots. A subsequent paralysis of the right ankle had supervened as the result of alcoholism. The case has been described in detail in the February number of the *Scottish Medical and Surgical Journal.*

(*d*) A woman showing the signs of a somewhat rapidly developing MUSCULAR ATROPHY of the Charcot-Marie type.

2. *Dr J. O. Affleck* exhibited a case exhibiting CHOREIFORM MOVEMENTS. The patient, a healthy man of 45, had suffered from these symptoms for seven years. A twelvemonth before they set in he had sustained a fracture of the leg, but it seemed doubtful whether this accident had had any connection with his ailment, as the symptoms appear to have come on gradually. There are constant choreic movements affecting the arms, legs, head, the muscles of expression, and to some

extent also those of articulation. They seem slightly slower in time than the movements of the chorea of childhood, but the difference is not great. They cease during sleep. The patient's mental condition is entirely unaffected, but he is precluded from his usual occupation, that of a gamekeeper, or from any other, by this condition of muscular unrest which is aggravated by any attempts at purposive movement. It was found that quietness reduced this choreic condition to a minimum, but beyond this all other treatment had proved useless.

3. *Dr Norman Walker* exhibited—

(*a*) A case of ACNE NECROTICA.

(*b*) Two cases of ICHTHYOSIS.

(*c*) A case of FAVUS, and several cases of LUPUS VULGARIS, which he had treated by exposure to the X-rays. The favus case was cured, and all the lupus cases were improved, some of them very remarkably. He referred incidentally to the method of using the rays, and to their advantages and risks.

4. *Dr Allan Jamieson* exhibited a case of LEPROSY. J. S., 17, born and brought up in India, of white parentage, and a healthy, well-grown lad. There were numerous lepers in the district where he lived, but, so far as he knew, he had not been in intimate association with any. There is a brown, smooth pigmentary stain near the spine, at the lower dorsal region, behind, but this has existed for many years, and is not anæsthetic. A year and eight months since, he noticed a small brown spot appear on the inner side of the left wrist, and this has gradually extended in area, till now it is the size of a half-crown piece. It causes no annoyance, and is the seat of no abnormal sensations. It is elevated about a quarter of an inch above the surface, feels firm on being pinched up, and, while brownish-red at the periphery and in the centre, is whitish over part of its area, and this white part is distinctly anæsthetic. There is no perceptible thickening of the ulnar nerve. Rather more than a month since, another spot showed itself at the margin of the axilla, on the right side; this is scarcely the size of a pea, and is not anæsthetic. He has had at times irregular feverish attacks, but whether malarial or leprous cannot be determined. With his consent, an oval wedge was excised from the central part of the patch on the

wrist; and this, after due preparation, was examined by Dr Lovell Gulland. He was not able, however, to discover any bacilli, and expressed the opinion that the sections had not quite the look of an ordinary leprous nodule, though there might have been leprous nodules in some parts at a former time. In tropho-neurotic leprosy bacilli are few in the cutaneous lesions, and this holds good also for the mixed form, to which, perhaps, the case may be held to belong. Taking all the features, there is no other disease than leprosy to which it can be allocated; but, so far, the type is a mild one. He has been ordered the natural salicylate of soda, in ten grain doses, twice a day, and, locally, an ointment of oxidised pyrogallic acid, salicylic acid, and ichthyol.

5. *Dr G. A. Gibson* exhibited a case of PSEUDO-BULBAR PARALYSIS, showing weakness of the muscles of the face and tongue, without any fibrillary movements or reaction of degeneration. The patient had interference with the voice and an altered cough, but very little trouble with swallowing. There were spastic symptoms connected with the limbs. The process was undoubtedly a chronic degenerative lesion of the motor area of the brain, with consecutive descending changes in the cord.

6. *Mr H. J. Stiles* exhibited (*a*) infant after operation for ACUTE ILIO-CÆCAL INTUSSUSCEPTION. Infant, æt. 5 months, admitted to Sick Children's Hospital at noon on Sunday, 19th November. Had had diarrhœa for a week. Twenty-four hours before admission vomiting set in, first of milk, then of bile-stained mucus, and six hours later the child passed two or three tablespoonfuls of blood-stained mucus. During the night more mucus was passed, and there was tenesmus, with paroxysms of abdominal pain and screaming.

One hour after admission to hospital the child was examined under chloroform. A firm, but somewhat ill-defined, sausage-shaped swelling was felt in the left half of the umbilical region, and it extended downwards towards the pelvis. The right iliac fossa and the right lumbar region felt abnormally empty. *Per rectum*, the lower end of an intussusceptum could be felt. No attempt was made to reduce the intussusception either by inflation or injection. The abdomen was at

once opened in the middle line above the umbilicus. The upper end of the intussusception was found a little below the left costal margin; the lower end reached far down into the pelvis. The incision was prolonged downwards below the umbilicus, and, with some difficulty, the lower end of the intussusception was brought out at the wound. Reduction was then effected by gradually squeezing upwards the lower end of the intussusceptum by manipulating it through the intussuscipiens.

Mr Stiles pointed out that, in reducing an intussusception, traction should not be made upon the entering tube. In this case there was no difficulty in reducing the invagination in the manner described.

The last portion of bowel reduced was the ileo-cæcal valve and vermiform appendix. The bowel was only slightly congested and œdematous; there was no peritonitis, but there was a slight tear in the peritoneal coat of the ascending colon, a little above the ileo-cæcal junction. After introducing two or three sutures of fine silk to repair the tear, the abdomen was closed. The child made an uninterrupted recovery, and left hospital upon the tenth day.

(*b*) Boy after Mikulicz's operation for DISEASE OF THE POSTERIOR TARSUS. Boy, æt. 7 years. Admitted to Children's Hospital September 19th. Three weeks before admission began to complain of severe pain in the ankle, and was very ill and feverish; the ankle became swollen and increasingly painful. On the fifth day of his illness pus was spontaneously evacuated from the outer aspect of the ankle. The skin in the neighbourhood sloughed, and the discharge became very foul.

On admission, the wound upon the outer side of the ankle was discharging freely, but the inflammation in the parts around had evidently subsided, and the boy's general condition gave no cause for anxiety. On the following day the foot was examined under chloroform, and on introducing the finger into the wound the astragalus and os calcis were found to be lying loose, and necrosed inside an abscess cavity, and could be hooked out. As permission for amputation had not been obtained, the operation introduced by Mikulicz for disease of the posterior tarsus was performed. There was hardly any bleeding from the distal part of the foot at the time of the operation, so that some anxiety was felt for its vitality. The following day, however, the toes were quite warm and natural. Five weeks ago

the patient was sent home with the limb in plaster. He has returned to have the plaster renewed, and the opportunity was taken of showing him to the Society, as cases suitable for treatment by Mikulicz's operation are rare. The limb is being kept in plaster in order that firm osseous union may be obtained before allowing the patient to walk upon the toes.

(*c*) Patient who suffered from ACUTE THYROID CACHEXIA after excision of a suppurating goitre. The patient, a female, aged 25, a native of Comrie, was sent to me for operation by by Dr H. A. Peddie. Her mother and two sisters all suffered from goitre. During the past three or four weeks the left lobe of the patient's goitre had become much larger, due, she said, to working in a draught. For several nights before the operation she felt feverish. When I saw her, for the first time, two days before the operation, I found that her temperature was 101·6. Both lobes of the thyroid were much enlarged, especially the left, the upper part of which was a little tender, and felt cystic. The tumour extended backwards beyond the posterior border of the sterno-mastoid, and downwards behind the manubrium sterni. She complained of breathlessness on the slightest exertion.

As the tumour was a large one, the angular incision of Kocher was employed, and the left sterno-laryngeal muscles divided. On attempting to separate the outer capsules from the goitre, an abscess, containing two or three ounces of pus, was opened into; it occupied the upper part of the left lobe. The wall of the abscess was composed of necrotic thyroid tissue. Other suppurating foci were met with lower down. In consequence of the adhesions between the outer and the true capsules of the thyroid, the "dislocation" of the latter was rendered much more troublesome than usual. In place of the isthmus, there was a third lobe, the size of a small orange, situated on the inner side of the base of the left lobe, and dipping down behind the sternum. This was not adherent, and shelled out without difficulty.

Two drains were introduced, one into the upper part of the wound, the other down behind the sternum, through a special opening between the two heads of the sterno-mastoid. The operation, although more severe than usual, was not followed by any shock to speak of.

Forty-eight hours after the operation the patient began to get very restless; she moaned constantly, and complained of

"pains all over." There was slight jaundice. The following day she lapsed into a state of extreme mental depression and lethargy, and did not seem to understand what was said to her. She refused food, and complained of pains in the legs; the toes were held pointed, and manipulation of the calf muscles seemed to set up painful contractions.

The lips were constantly being firmly pressed together, and twitchings of the face, especially about the angles of the mouth, were frequently noticed. The wound was dressed, but nothing was discovered to account for the symptoms: there was no hæmorrhage; the upper drain was removed. I had no doubt but that the symptoms pointed to a condition of acute thyroid cachexia, due either to a diseased condition of the remaining right lobe, which was, therefore, unable to carry on the normal thyroid functions, or to the absorption into the system of abnormal juices set free from the diseased lobe during its removal. Seventy-two hours after the operation the patient was given thyroid tabloids (B. & W.) in five-grain doses every four hours. After the second dose a great improvement was noticed in the patient's general condition; twenty-four hours after the commencement of the administration all mental torpor had disappeared, as also had the cramp-like pains in the legs and the twitching of the facial muscles, and the patient expressed a desire for food. The temperature, which had been subnormal, rose to between 99° and 100°. The patient made a rapid and complete recovery, and returned home with the wound completely healed at the end of the third week. She now takes one five-grain thyroid tabloid night and morning.

(*d*) Patient, after excision of a large (simple or malignant?) LYMPHOMA OF THE NECK. The patient, a male aged 26 years, was operated upon on October 1st for a large glandular tumour of the neck. The swelling extended from the angle of the jaw almost to the clavicle, forwards to the middle line, and backwards under the sterno-mastoid into the posterior triangle. The main part of the tumour, which was smooth and about the size of a lemon, was situated in the carotid triangle; around it the swelling was nodular, and evidently made up of a mass of enlarged lymphatic glands. There were no signs of softening in any part of the swelling. The large, rounded mass was firm and somewhat elastic, but the other glands had a more doughy feel.

The swelling appeared suddenly four months previously and had steadily increased. It was never painful, but the patient's general health had deteriorated. The diagnosis rested between tubercular adenitis and lympho-sarcoma. A cautious prognosis was given.

The operation was an extensive one, and necessitated division of the sterno-mastoid. The glands were not particularly adherent, but they were very soft and friable. On section they had the uniform greyish-pink, fleshy, and somewhat medullary appearance, characteristic of a round-celled sarcoma. Moreover, the wound healed with a rapidity characteristic of sarcoma. The microscope showed a structure typical of lympho-sarcoma. The patient, who was engaged, was advised not to think of marrying in the meantime. Mr Stiles looked upon the condition as lympho-sarcoma, and requested the patient to report himself at short intervals. Three months have elapsed since the operation. The patient's general health has improved, and there is no trace of any recurrence. Mr Stiles considered the case of interest as showing that, both clinically and microscopically, it is sometimes impossible to distinguish between simple lymphoma and lympho-sarcoma. Our knowledge of the exact nature and etiology of these lymphomatous tumours leaves much to be desired.

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(*e*) Patient, after AVULSION OF THE ENTIRE SCALP. The patient, a girl aged 12 years, was sent to the Children's Hospital (by Dr Connell, of Peebles) on May 3rd, nine weeks after the accident. While skipping in a public laundry, first the rope, and then her hair, was caught by a revolving wheel, which stripped off her entire scalp, including the upper portions of both ears.

On admission, the patient was very weak. The raw surface of the scalp was covered by very vascular and œdematous granulations, except over the left parietal region, where there was an area, about 2 inches square, exposing dead and parchment-like pericranium. After removal of the latter, the subadjacent bone gradually became covered by exuberant granulations. No necrosis or exfoliation has occurred.

Six weeks after admission, the granulations seemed to be sufficiently healthy to admit of skin-grafting. This was done, upon two occasions, in the month of June, skin having been obtained from two students attending the cliniques. On both

occasions the grafts took well, but subsequently became devoured, apparently by the very vascular and hypertrophied granulations; so that, about four weeks after grafting, they had entirely disappeared. Skin from a large number of circumcisions was from time to time planted, but it suffered the same fate. The patient was sent home in the end of October, in the hope that a change to the country would improve her general health. While at home, she unfortunately suffered from an attack of erysipelas. She was re-admitted a fortnight ago. On the next occasion, Mr Stiles said that he intended to remove the granulations before applying the grafts, which will be taken from her own skin.

7. *Dr F. D. Boyd* exhibited a patient with signs and symptoms of SYRINGOMYELIA. The patient, a man aged 50, was admitted to the Deaconess Hospital, complaining of "pain in the right side of the chest, headache, loss of sensation in the left hand and arm, and hoarseness." On examination, it was found that the patient, a well-built, rather youthful-looking man, showed some interesting lesions.

The *sensory functions* showed: Headache, pain in the back of the neck, especially on the right side, and pain and tenderness in the right infra-clavicular region. Tingling in the fifth finger of the right hand, extending up the ulnar side of the forearm to the elbow. Numbness in the left hand, both back and front, as far up as the wrist. Sensation of cold in the left foot. Intelligence good. Memory fair. Sleep good. Speech normal, save for the alteration in the voice.

Examination of the cranial nerves good. Sense of smell impaired. Eyesight, pupils react normally. Refraction normal. Full astral vision. No optic atrophy. Concentric contraction of field of vision in the right eye, as also in colour field in left eye field of vision, normal. Movements of eyeballs good and equal. No nystagmus. Sensation in area of fifth nerve good. Seventh nerve normal. Hearing diminished on both sides, especially the right. Sensation at the back of the mouth was slightly hyperæsthetic. Tongue protruded quite straight, and movements good. There was some paresis of the trapezius.

Motor functions.—The grasp of the left hand was weak. Abduction and adduction of the fingers of the left hand was considerably impaired. The right trapezius was paretic, and

the shoulder could not be raised properly. The left pectral and both the deltoids showed diminished power.

Co-ordination in the left hand was impaired; normal in the right and in the legs. Nutritive functions were impaired at parts. There is marked atrophy of the thenar eminence on the left side. Slight atrophy of the hypothenar. No visible interosseous atrophy. The left pectorals, the deltoids, and the right trapezius were atrophic. The reflexes were normal.

Sensory functions showed diminution or loss of sensation in the palmar aspect of the left fingers and hand, diminished sensation in the dorsal aspect of the hand, with the exception of the last phalanx of the thumb. Over the rest of the body sensation to touch normal.

Sensation to pain normal except over the palmar aspect of all the fingers of the left hand and over the dorsal aspect of the last phalanx of the thumb where the prick of a pin was felt as a touch. Over the rest of the dorsal aspect of the hand and fingers there was no sensation felt on the prick of a pin.

The temperature sense was impaired or lost over parts of the left hand and fingers and the plantar aspect of the toes.

Electrical reaction of the muscles showed normal reaction to the faradic current.

With the galvanic current normal polar reaction was got in all the muscles except the left abductor brevis, which did not react. In the muscles of the left hypothenar eminence and the right trapezius a much stronger current was required to elicit reaction than on the opposite side.

The respiratory system showed paralysis of adduction in the right vocal cord.

There was paralysis of the right side of the palate.

Over the upper part of the right side of the thorax there is impairment of percussion, with feebleness of the breath sounds.

(*b*) A patient with DOUBLE FACIAL PARALYSIS OF PERIPHERAL ORIGIN. The patient, a man 32 years of age, was admitted to the Deaconess Hospital suffering from alcoholic peripheral neuritis, with the usual symptoms and signs in the legs and arms, but accompanied with paralysis of the left facial nerve and pain over the course of the nerve. Three days after admission pain was complained of in the right side of the face, and on the following day paralysis of the right facial had developed. The face, which had previously been distorted,

became symmetrical from the double paralysis. The patient could shut neither eye. There was some motor paresis of the fifth pair of nerves, but no implication of sensation.

Under treatment the right facial recovered fairly rapidly, but the left remained affected.

(*c*) A patient, 32 years of age, who illustrated how complete a recovery was possible in PERIPHERAL NEURITIS, even when the toxic agent affected the central nervous system. In November 1898 the patient suffered from the vomiting of pregnancy, which was so severe as to reduce her to a state of extreme emaciation, with in addition a bed sore. After confinement she improved rapidly for a time, but then began to complain of loss of power and numbness in the legs and arms. There was no evidence of sepsis at the confinement.

On admission to the Deaconess Hospital, in April 1898, the patient was found extremely emaciated. There was complete paralysis of the legs, with foot-drop, paralysis of the arms, loss of sensation in the legs below the knees, reaction of degeneration in some muscles, entire absence of electrical reaction in others. The organic reflexes were abolished. The mental and moral functions deteriorated with loss of memory.

Under treatment the patient entirely recovered. The muscles are now active. The memory has returned, with the exception of the memory for events during her illness. The moral nature and whole aspect of the patient has changed for the better.

II. Exhibition of Specimens

1. *Dr A. C. Sym* exhibited a specimen from a case of MORBUS LUDOVICI on account of its rarity, the *post-mortem* examination having been done by Sir Henry Littlejohn and Dr Harvey Littlejohn. Dr Sym was called in only two hours after the onset of severe symptoms, though for a week the patient had complained of sore throat. On his arrival, Dr Sym found the case one of dyspnœa and cyanosis, and gave ether hypodermically with no effect, as the patient died within an hour of being seen. There were no indications for tracheotomy. The pathological appearance was one of extensive sloughing phagydœna involving trachea pharynx and the whole of the tissues of the neck. The lungs were not involved.

2. *Mr J. M. Cotterill* exhibited (*a*) a SARCOMA OF THE BRAIN, about the size of a tangerine orange, removed from the right motor area of a gentleman aged 40.

Symptoms of involvement of the leg and arm centres had existed for about eighteen months; and there had been several epileptiform attacks, which of late had become somewhat worse.

The tumour was distinctly encapsulated, and appeared to spring from the falx cerebri, opposite the top of the fissure of Rolando. Bleeding was slight, and the patient made an uninterrupted and complete recovery.

(*b*) An INTRAMUSCULAR FATTY TUMOUR, of about the size of a cocoanut, removed from inside the substance of the triceps muscle in a man 62 years of age.

The interest of this case lies in the great rarity of *intra*-muscular as contrasted with *inter*-muscular lipoma, the latter condition being quite common.

(*c*) A very large URETHRAL CALCULUS (uric acid) 3 inches long and 2½ inches in diameter, removed from the urethra of a middle-aged man (sent by Dr Reid of Kirkcudbright).

3. *Dr G. A. Gibson* exhibited a specimen of a HEART showing a characteristic patch of myomalacia caused by thrombosis of a branch of the anterior coronary artery. Sections from the softened patch were exhibited under the microscope. They showed granular changes in the muscle cells, hæmorrhages into the intermuscular tissues, and some newly-formed fibrous tissue.

4. *Dr R. B. Purves* exhibited a specimen of EXOSTOSIS OF FEMUR removed from a man of 48, whose symptoms commenced three years ago. When pulling up a bush he fell backward, and on rising felt that he had hurt his left thigh. Was painful, and began to swell. Lasted thus for three months. Pain and swelling gradually subsided, leaving left thigh rather thicker than right. Pain easily recurred when he did heavy work. Occasionally, when pain came on, by moving his leg he felt something slip in his buttock, and this eased the pain, as a rule. Family and previous history negative.

Two days before admission pain came on suddenly while he

was walking. Was giddy and sick. Thigh more swollen, and painful and tender. Swelling was chiefly to the outer side, and was in part tense and fluctuating. Was tapped, and a clear semi-gelatinous brownish fluid obtained; about 25 ozs. (Nothing found in it by microscope like tumour cells.)

Then a hard mass was felt high up on femur below trochanter and toward posterior aspect. Hard, irregular, no crackling.

On incision found a large adventitious bursa which had formed over an exostosis growing from posterior aspect of femur below trochanter. Removed and drained.

Meeting IV.—January 17th, 1900

Mr A. G. MILLER, *President, in the Chair*

I. ELECTION OF MEMBER

Charles Begg, M.B., C.M., Bridge of Allan, was elected an Ordinary Member of the Society.

II. EXHIBITION OF PATIENTS

1. *Dr Norman Walker* exhibited a case of ACNE VARIOLIFORMIS, which formed an interesting contrast to the case of acne necrotica which he showed at the previous meeting. The patient was a man aged 35, and had suffered for two or three years. The forehead, adjacent portion of the scalp, and the temples, were affected. The patient denied all history of syphilis, and no traces of the disease could be found. The spots were raised, red, somewhat painful on pressure; the condition was distinctly inflammatory, and there was no sign of necrosis. Each lesion left a deep scar, and it was from these scars that the descriptive adjective was derived, the "acne" spots were not varioliform. The patient, like most of the previously described cases, improved slightly on iodide.

2. *Dr C. W. MacGillivray* exhibited a patient who had suffered from a rare form of FRACTURE OF THE SKULL.

The patient, a boy aged 15, was struck on the left parietal region by a large iron gas-pipe, which fell a distance of 30 feet. He sustained a severe semilunar lacerated wound of the scalp, with a compound fracture of the skull, a portion of the left parietal bone the size of an oyster shell, and comprising the whole thickness of the bone, being found lying loose in the wound, being entirely separated from both the pericranium and dura mater. This was removed, and the depressed upper edge of the gap trephined. Beyond having been stunned at the time of the accident, he presented no symptoms, and made a perfect recovery.

III. Original Communications

1. CLINICAL EXPERIENCE WITH SALOL IN TREATMENT OF SMALL-POX

By Charles Begg, M.B., C.M., Bridge of Allan

THE following short statement of my experience in China with Salol is of interest at the present time, seeing that we are unfortunately dealing with an epidemic of small-pox in Hull and may at any moment be face to face with it elsewhere.

In speaking of Salol I pass over all reference to its use in intestinal cases, merely recording my strong conviction of its great value.

An observation I made during the progress of such a case led me to believe that it might be useful in quite another class of cases, and it is this I wish to call attention to to-night, not being aware of similar observations having been recorded.

My patient was taking four powders per day, each containing 15 grains Salol. They produced a condition of slight mental depression, but the point which struck me was, that although he was, when in health, specially liable to severe irritation from mosquito bites, he now suffered no discomfort from their attacks and hardly noticed their attentions. This was not due to the mental condition. The puncture from the bite was plainly visible with a total absence of the characteristic reaction, neither swelling, heat, nor irritation being present.

I followed up this observation by giving the drug freely in cases of irritable skin diseases, acute eczema, prurigo, etc. of which we see many cases in the Tropics, and which on account of climate are far more trying to the patient and more urgently call for relief. In these cases I had my belief in the power of the drug confirmed. Then naturally I thought of small-pox, and in trying it obtained remarkable results. First, it kept the patient free from all sense of irritation without the slightest desire to scratch. Those who know anything of small-pox cases will at once see the great gain to the patient, preventing exhaustion from sleepless nights and drain to the system from weeping vesicles and pustules torn by scratching, avoiding danger to the eyes from soiled fingers, conserving energy for the most trying time of the disease, namely, the convalescence. But further, I found in the second place that almost all the vesicles terminated as vesicles, and the stage of maturation was absent except in a few isolated vesicles which matured in the usual way. In my last case, an Englishman, aged 60, with confluent small-pox, only two groups of vesicles, one on either upper arm, matured; they did so with perhaps greater energy than usual, taking on more the character of abscesses. In all the other vesicles the lymph dried up and there was an end of them. The relief to itching was most marked and could be demonstrated to depend on the drug, returning at once on reducing the dose.

I have not observed any evil effects from a daily dose of ʒi continued for long periods, and the mental depression quickly passes off with discontinuance of the drug, nor does it always appear in any marked degree.

Salol is accused of having caused death from 120 grs. given during eight hours. The death took place four days after. One would therefore like to know more particulars.

Salol is said to be of slow excretion, but allowed to be quickly decomposed into its component parts by the whole digestive tract except the stomach.

Both the phenol and salicylic acid are found abundantly in the urine, which becomes dark; but a large quantity must be given off by lungs and skin, because the characteristic sweet, fruity odour is very quickly perceptible in the room used by the patient, and is more pronounced than in the case of any other drug I know.

In acting as an intestinal disinfectant, it is acting, of course, quite outside the body; but the marked affinity it shows for elimination by lungs and skin would explain the above results of clinical observation by allowing it to act directly on the nerve terminals, and to reach the vesicles more surely than one could apply it with our hypodermic needle, a treatment long ago suggested in the case of abscesses by carbolic.

I have just found out that Salophen has been reported in the medical papers for 1897 as of use in pruritis, which would go to confirm my observations. *Salophen* was introduced to replace Salol on account of the ill effects thought to result from the elimination of phenol. It is said to quickly remove the pain and swelling in acute rheumatism, but does not prevent relapses.

I trust that those of you who may have the opportunity will not fail to bring your small-pox cases early and fully under the influence of this drug, in order that we may see if the results obtained in China are confirmed here. I know of no reason why it should not be so, for it is during the cool season in China the disease shows itself. Still there is always the charge to be met, namely, that one has perhaps been dealing with a milder type of the disease, or that the drug has been helped in its action by climate. Just as in my treatment of sprue, until I successfully treated cases from India, it was doubted whether the disease as seen in China was at best more than a mild form.

2. PERMANENT DISLOCATION OF THE PATELLA

By JOHN SHAW M'LAREN, M.A., M.B., F.R.C.S.Ed., Assistant-Surgeon, Royal Infirmary, Edinburgh; Lecturer on Surgery, School of the Royal Colleges, Edinburgh

APPEL of Munich, in an interesting paper published some years ago, has given the result of his study of this rare condition. A case recently under my care, and a study of some others not mentioned by Appel, enable me to support the views which he urges with regard to the nature of the dislocation, and to indicate the best method of treatment, a matter which he does not touch.

The dislocation is a displacement of the patella outwards,

so that it lies, even in extension of the knee, in most cases to the outside of the middle line, and in flexion it slips outwards and backwards till it lies on the outer surface of the external condyle of the femur, its anterior surface directed partly outwards; the patient's gait is quite insecure, and frequent falls and synovitis of the knee are the result.

I believe that, in most cases, a fall, and consequent injury to the knee, first direct attention to the condition, and indeed may increase the dislocation to a certain extent; but the dislocation really depends, as Appel holds, on a congenital defect, namely, a deformity of the external condyle and trochlea of the femur. The reasons for this view will appear.

In most of the cases recorded, one or other of the following conditions will be found:—(1) The dislocation, or some accompanying deformity actually noticed at birth; (2) the late age at which the child learnt to walk, or else insecurity in walking during childhood; (3) rachitis; (4) genu valgum; (5) outward rotation of the leg; (6) ill-developed patella; (7) abnormality in the shape of the external condyle and patellar fossa of the femur.

With regard to my own case, I have noted: J. M., age 19, domestic servant, admitted to hospital May 29th, 1895. Her mother states that she never walked securely like other children, and in fact could not be said to walk much at all till she was seven years of age; that she was never able to run, and was constantly falling; that she became knock-kneed, and that, while the right knee improved (so she said), the left remained deformed. Three months ago she fell going upstairs, "her leg seemed to give under her, and she fell quite helplessly." She had fallen frequently since in the same manner, and had been laid up in consequence more than once with synovitis of the knee.

She is a healthy-looking girl, but with evidence of rachitis in childhood; very short and clumsy figure. There is marked genu valgum on the left side; the leg is rotated outwards. When the knee is extended, the patella lies somewhat to the outside of the normal position; when it is flexed, it passes further and further out till it lies on the outer side of the external condyle of the femur, its anterior surface directed outwards and forwards. It may with force be held in or near the middle line while the knee is being flexed to a certain extent,

but, on being released, it flies out and back. The fossa patellæ of the trochlear surface of the femur can be felt to be partially filled up, as it were, on its outer side, as if the external condyle projected into it; while the outer edge of that condyle, on the other hand, seems to fail altogether, and leave a gap under the outer edge of the patella. The Röntgen rays show the patella to be higher than normal, and give a faint indication of the altered shape of the external condyle.

Appel, including two cases of his own, gives thirty cases. Of these, unfortunately, in twelve no mention is made of the condition of the femur; of the remaining eighteen, fifteen had a deformity of the femur, and in three it was said to be normal. The notes of Appel's two cases are as follows:—(i) A woman, aged 40; as a child, late in learning to walk; nine weeks before admission to hospital, fell in the harvest-field and injured her knee; since then complained of weakness in standing, with the knees flexed, especially if the trunk was held backwards, as in descending a stair. Patella smaller and rounder than normal. It slips back outside the external condyle on flexion. Groove on the femur for the patella shallow and narrow, due to the edge of the trochlea being broad and rounded, its normal sharp ridge lost; outer side of the external condyle slopes away, instead of falling away sharply. Slight genu valgum. Leg rotated outwards. (ii) A man, age 26; "double-knee" remarked at birth; at work as a porter felt his left knee weak on descending a hill with heavy weights on his back. The patella lay on the external condyle, and slipped out and back on flexion of the knee. Groove for patella narrow, due to a mass of bone projecting from the external condyle. Slight genu valgum. Leg rotated outwards. The same characters appear in one or other of the cases collected by Appel. Thus in Uhde's case the fossa patellæ was too narrow, and the ridge of the external condyle bounding it was not so prominent as usual. Stokes, Smith, and Servier each records a case in which the abnormality is described as an absence of the external condyle. Lannelongue mentions a very small patella and a diminished trochlear surface for it. Janicke records smallness of the external condyle, shallowness of the fossa patellæ, and rickets.

I have collected some cases not recorded by Appel. Wright records of his patient, a girl 7 years old, that she did not

walk till she was 3 years of age; some months before he saw her she had fallen while dancing, and had been constantly falling since; the patella was smaller than normal, but he describes the external condyle as "very prominent," possibly referring to its invading the fossa patellæ. There was the usual dislocation of the patella. In Pollard's case the patient, a woman aged 21, could not fix the period at which her patella was displaced, but the limb had been "weak since childhood." A few weeks before being examined she slipped and fell, and had been lame since; there was marked genu valgum, the patella was smaller than usual, and lay on the outer side of the external condyle during flexion of the knee; when he operated he found the trochlear surface of the femur "exceedingly small in all its dimensions," and the groove for the patella very narrow. In Goldthwait's case the patient was a woman 30 years of age; both patellæ were dislocated, and the deformity had been noticed since she was a child; the leg was markedly rotated outwards. In Canton's case (just mentioned in Appel's list) there was the usual dislocation of the patella, knock-knee was present, and the external condyle "did not project anteriorly to its wonted degree." He continues quaintly—reversing apparently the cause and the effect—"the dislocation of the bone was noticed at birth, but was not believed to be of any importance; and now the external condyle has become so worn away that the case is beyond the reach of surgical art."

It has been pointed out that the quadriceps in passing from its origin to its insertion forms an obtuse angle at the point of which the patella lies. In action it would draw the patella outwards till it came to lie in a straight line between the origin and insertion of the muscle were this not prevented by the obstacle which the prominence of the external condyle presents. The ridge of the external condyle keeps the patella in the middle line. Should the fossa patellæ be too shallow, or the ridge of the external condyle not be sharp enough, the patella will lie dangerously far out on the trochlea—a case of slight dislocation. It thus only needs a severe contraction of the muscle or a slight trauma to shoot the patella off the front of the condyle altogether, and bring it to lie on the outside of the femur. Or in cases in which the condyle is markedly deficient the complete dislocation may exist from birth or the first years

of life. In any case the structures on the outer side of the patella eventually shorten and prevent the possibility of the bone retaining its proper position. It is natural to ask the question, What is the connection between rickets and this dislocation, but it is not easy to give an answer. In some cases it is possible that both the knock-knee and the outward rotation of the tibia are *produced by* the abnormal line in which the quadriceps comes to work.

As regards the treatment of this condition, we find, as we might expect, various methods adopted. Roux (in a traumatic case) divided the vastus externus, detached the ligamentum patellæ, and nailed it further inwards to the tibia. Goldthwait in his bi-lateral case adopted a similar procedure in treating the one knee, while, on the other side, he detached the tubercle of the tibia and nailed it further inwards; he gives the preference to the latter method. On both sides he divided the outer part of the capsule to allow the patella to be drawn inwards. The result was good. Lucas Championière performed a more severe operation. He opened the joint, cut a groove on the surface of the inner condyle of the femur, and fixed the patella in it by sutures. Bilton Pollard followed this idea. He corrected the genu valgum by osteotomy, but the patient's walk was no more secure. To correct the dislocation he made first an external incision through which he divided the insertion of the vastus externus and the outer part of the capsule; through an internal incision he chiselled out a groove in the cartilage and bone of the femur "till a sufficiently broad and deep trochlear surface was made." The patella placed in this slid up and down normally in flexion and extension of the knee. Lastly, he excised a piece from the inner side of the capsule, and braced up the joint. Result—after a month the patient walked well, and said her knee was stronger and more serviceable than before the operation. Wright adopted a much simpler procedure, and one that was followed by a good result; it resembles the operation I performed, which I shall now describe.

I had not seen any record of similar cases, nor of the methods of treatment, but, if I had, I do not think that I should have preferred any of them to that which suggested itself to me. It consisted in liberating the patella by dividing on the outside whatever contracted structures prevented its assuming

its normal position, and then mooring it there by sutures passed through its inner border.

On June 8th, 1895, I corrected the knock-knee by an osteotomy, but, when the patient began after the usual period of rest to go about again, she complained still more of insecurity in walking—possibly due to some atrophy of the quadriceps.

On September 3rd I made a U-shaped incision over the knee, and turned up a flap of skin and fat. I divided on the outer side of the patella the expansion of the quadriceps tendon and the capsule, but without opening the cavity of the joint, so as to allow the patella to be brought easily to the middle line; I then bored two holes through its inner edge, and having passed a stout catgut suture through each, I fixed the patella in its place by stitching it to the internal lateral ligament. The wound healed by first intention, but the sutures were not strong enough, for I had the annoyance of seeing the patella gradually slip back to its old position.

About eight weeks later I performed the operation again, precisely as before, using this time, however, stout silk instead of catgut sutures. There was again an uninterrupted recovery, and the patella remained in its normal position. The operation was followed by a most marked and prolonged atrophy of the quadriceps and corresponding uselessness of the limb. For a long period afterwards, if she were lying down, she could raise her heel off the couch only if the knee-joint were extended. The limb, however, under massage, electricity, and exercise, gradually gained strength, though for months she had to walk with care. On November 30th, 1899, four years after the operation, I examined the knee. The patella had retained its position, somewhat external to and higher up than the normal, and the patient walks, and goes up and down stairs with confidence, having lost all fear of falling. Wishing further to test her, I asked her if she could *run*. She replied with a laugh that recently she had won the third prize in a race at a picnic.

This method, then, of treatment—dividing contractions on the outer side, and stitching the patella by sutures passed through the structures on the inner side—seems rational and effective, and should succeed in most cases. It is possible, however, that the severer procedure of opening the joint and enlarging the groove for the patella might in some cases be necessary; while, again, if the outward rotation of the leg is very marked, probably

it would be wise to detach the ligamentum patellæ and fix it further inwards.

3. A CASE OF MURDER AND SUICIDE

By HARVEY LITTLEJOHN, M.A., M.B., B.Sc., F.R.C.S.Ed.,
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WHEN two bodies are found in the same room with fatal injuries upon them, several questions of medico-legal interest and importance arise. Amongst these, one of the first requiring an answer is whether we have to deal with death from accident, suicide or murder, or a combination of two of these conditions.

The accidental death of two persons together as a result of external violence is as a rule recognised without difficulty from the surroundings and nature of the injuries, the cause of death being usually obvious, and all the circumstances pointing to a sudden and unexpected calamity.

The question of murder or suicide is not always so easily determined, and a decision upon this point can, in most instances, only be arrived at after careful consideration of the whole circumstances and an examination of the injuries.

By the circumstances are meant chiefly those connected with the position and surroundings of the bodies, and it is of the highest importance that the medical man, who is required to give an opinion in such cases, should have the opportunity of making his examination of the bodies and the immediate surroundings before they are in any way interfered with.

The position of a body may be wholly inconsistent with a theory of suicide, or on the other hand, it may afford strong presumptive evidence of it, and in the same way the position of any weapon found or of marks of blood on furniture or in the room may afford circumstantial evidence of the strongest character, leading to the discovery of an unsuspected crime. It is obvious that where a decision on so vital a point as the determination of the question of murder or suicide may rest largely in certain cases upon such circumstances as the position and surroundings of a body, and more especially in cases where two bodies are involved, no interference with them should take place until an examination has been made by a competent authority.

Many points which to the police officer appear to have no significance in the case may be of the greatest importance and assistance to the medical examiner in arriving at a correct interpretation of the case.

When once the position of a body or weapon has been altered the difficulty of exactly reproducing in every detail the original arrangement is well known, even when careful observations have been taken, but naturally it becomes well nigh an impossibility when the interference has been carried out hastily and by ignorant persons.

The following case which occurred some time ago, and which I had an opportunity of observing, illustrates the above points and also some other questions of medico-legal interest. One morning a gentleman arrived at an hotel and engaged a room for himself and his wife, who he stated was to join him later in the day. His appearance and manner attracted no particular attention, and he employed himself during the afternoon in writing letters in his room. In the evening he went out and returned to the hotel accompanied by a lady, and after having some refreshment, they retired for the night. Next morning at breakfast, they both appeared to be perfectly happy, and on good terms. They went out apparently to do some shopping and returned to the hotel about one o'clock and immediately proceeded to their bedroom. Nothing peculiar was noticed about either of them except that the man was muttering to himself and that he slammed the door of the bedroom behind him.

Nothing more was seen or heard of the couple, although, their luggage having been packed, it was presumed that they intended to leave that afternoon, until seven o'clock in the evening, when an entrance was forced into the room and they were found lying upon the bed in a pool of blood.

Both were fully dressed just as they had been when out walking, with the exception of their hats. The room was in no way disordered, and there were no marks of blood anywhere except upon the bed. The body of the woman lay on its back across the bed, her legs hanging over the edge so that the feet almost touched the ground. It was warm, but rigidity of the neck and arms had already set in. Her face, which was turned towards her right shoulder, was covered with blood. The right arm was semi-flexed and extended from her side so that it

lay on a level with her head. The hand was covered with a tightly-fitting strong dogskin glove, which showed no trace of any cuts or marks of blood. The left hand, which was also gloved, lay by her side under the body of the man, which extended transversely across her. Her dress showed no disarrangement.

In the neck on the left side there was a wound, with clean regular edges, four and a half inches long and extending from half an inch below the symphysis menti downwards and backwards to a point about two inches below the angle of the lower jaw. All the structures were divided down to the large vessels upon the left side—the internal jugular vein being completely cut through, while the common carotid artery was laid bare but was uninjured. The thyroid cartilage was completely divided just above the true vocal chords, and the incision passed backwards through the posterior pharyngeal wall to the vertebral column, which was deeply scored. The large vessels upon the right side were exposed, but were uninjured. There was no evidence of any second attempt at dividing the structures, all were cleanly cut through by a single gash, even the thyroid cartilage exhibiting a perfectly regular and even line of division. The injuries to the structures on the right side of the neck and the complete division of the larynx are peculiar in view of the fact that the wound in the skin did not extend beyond the middle line of the neck, but I account for the nature of these injuries by the head having been forcibly turned towards the right shoulder, and to the force having been directed obliquely backwards from the left to the right side of the neck—the wound was thus much deeper towards the right side than on the left, where it tailed off in the superficial subcutaneous tissues.

The deceased had on a jacket with a high collar, and the upper margin of this collar had been cut through on the left side opposite the wound in the neck.

A point of interest and importance was the presence of two superficial wounds in the skin of the face, one on the left side of the nose, and the other on the middle of the forehead. Both presented the appearance of having been inflicted by a very sharp knife, and were merely superficial, running obliquely into the skin from the left to the right side, and evidently of quite recent origin. There was no blood on the front of her dress or upon her hands and arms, but there was a considerable

quantity on her face and on either side of the neck, down which it had flowed, and deeply stained the bed-clothes immediately beneath those parts.

The man lay extended upon his face across the body of the woman, his head resting upon the pillow and his feet at the foot of the bed. Both arms were extended from the side of the body in a semi-flexed position, and close to his right hand, which was covered with blood, there was an open razor likewise covered with blood. His throat was cut from "ear to ear," the thyroid cartilage being completely divided just below its upper border, and from another deep gash in it a little lower down it was evident that more than one attempt had been made to divide it. All the structures down to the deep vessels on either side were divided, but only the internal jugular vein upon the left side, among the large vessels of the neck, was injured. The pharynx was not opened into. At the commencement of the incision in the skin upon the left side there were several of those superficial cuts so frequently seen in cases of suicide, while in the woman this appearance was absent.

The front of his body was drenched with blood. The blood marks on the bed, however, were confined to those portions of the bed immediately under the bodies, the rest of the covering white counterpane being absolutely spotless. This is interesting, and corresponds with what we should expect from the nature of the injuries found, viz., in both cases wounds of the jugular veins but not of the larger arterial trunks, while it also indicated that neither of the two deceased persons moved from the bed after the infliction of the injuries.

In the pockets of the man's coat there were found some rings belonging to the woman, two razors, and two letters foreshadowing the death of both of them, but indicating that it was to take place by common consent. These were probably the letters which he was seen to be writing the previous day, as they bore that date.

From the above account it is evident that we have to deal with injuries produced either homicidally or suicidally. There are three possibilities which have to be considered, *first*, that both were murdered, *second*, that both committed suicide, or *third*, that one murdered the other and then committed suicide.

Against the first of these suppositions many points may be urged, and there is little difficulty in disposing of it. The fact

that the door of the bedroom was locked upon the inside, and that there was no access by means of the window; further, the fact of no cries being heard, or of any signs of a struggle being present in the room, or on the persons of the deceased, such as one would expect in a homicidal attack upon two active adults, all strongly negative the possibility of a double murder. The character of the wound of the throat in the man's case, but more especially the distribution of the blood in the room, viz., its being confined to the bed and those parts of it immediately underneath the bodies, do not favour such a possibility. In regard to the second supposition—that of double suicide—it is well known that two people or even a larger number sometimes decide to end their lives together by their own hands, and this possibility is all the more probable when we have to deal with two persons of different sex. Domestic difficulties and affliction or a hopeless attachment are sufficient inducements for some people to end their existence, and the possibility of such circumstances having existed in the present instance must not be lost sight of. On the other hand, it is very rare that such a double suicide has been effected by cut throat. Women, and more especially women of some refinement, as in the present case, do not care for methods of self-destruction which entail severe injury and disfigurement of the person: their inclinations tend rather to such means as drowning, hanging, and poison, by which methods death is known to be comparatively painless and yet to be equally certain to result.

From the mode of death, therefore, in this instance, the probability is against the suicide of the woman, while her whole bearing as observed by several witnesses, and the fact of there being no other indication of her intention, as was the case with her companion, as his letters testify, render this probability yet stronger.

In addition to these considerations, the character of the wound upon the neck, its position, its direction from above downwards and backwards, and from right to left, together with the fact of her hands being enclosed in tight strong leather gloves which must have interfered with the power of the hand to inflict such a severe wound, the absence of blood on the gloves and down the front of her dress, entirely exclude, in my opinion, the theory of suicide on her part.

There remains, therefore, for consideration only the third

proposition, viz., that the case was one of murder and suicide. From what has been already stated, and looking to the fact that we have to deal with a strongly built man and a comparatively weak woman, practically the only possibility is that the man was the murderer, and afterwards committed suicide. This view is borne out by all the circumstances.

The woman, as previously stated, was fully dressed even to her outdoor jacket and gloves, and the former had actually been cut through in the production of the wound in the neck. This circumstance alone is of great significance, since a suicide by cut throat always take care that nothing intervenes to prevent the easy accomplishment of the deed. The wound itself, commencing below the chin, and running from above downwards and backwards on the left side of the neck, is a wound which might be self-inflicted by an ambidextrous or left-handed person, but is wholly inconsistent with self-infliction by a right-handed individual.

At the same time, I may remark that a very considerable experience in cases of cut throat has brought very forcibly before me the fact that the descriptions given in many books on medical jurisprudence, of the distinctive characters of suicidal and homicidal cut throat wounds, as regards their situation and direction, must be accepted with caution, inasmuch as suicidal wounds may be situated practically anywhere in the neck, and present the appearance usually stated to be characteristic of a homicidal wound.

The depth and extent of a wound in the throat afford little help in making the distinction, since a suicide is able not only to divide the larynx and soft parts as far back as the vertebral column, but, as in one instance which came under my observation, he may divide an intervertebral disc, and even penetrate to the vertebral canal.

In the present instance of the woman, however, it is rather the direction of the wound in a right-handed person than the depth or severity which, in my opinion, excludes suicide, so far as the injuries are concerned. In a homicidal attack with a knife one would certainly expect the defendant to exhibit some signs of resistance, more especially upon the hands, where cuts from attempts to grasp the knife are very characteristic. Such injuries are wanting in the present instance, with the exception of the two small cuts upon the nose and forehead, which

were probably produced by some slight struggling and movement of the head, and in all probability the absence of other signs of a struggle is to be accounted for by the attack having been made suddenly while the defendant was lying upon her back, and without any warning of her assailant's murderous intention.

As strongly as the evidence in the case of the woman points to homicide, so does that in the case of the man indicate suicide. The position, direction, and extent of the wound in the throat, the razor found on the bed near his right hand, the blood upon his right arm and hand and on his clothes anteriorly, all indicate suicide, while the respective position of the bodies points to the deed, in the man's case, having been executed subsequent to the infliction of the injuries to the woman.

If one may attempt to picture the course of events, I believe that what occurred in all probability was the following: When they returned from their walk, and shortly after they entered the bedroom, the woman was sitting on the edge of the bed. The man, probably quite playfully, pushed her backwards, and kneeling over her, suddenly took a razor from his pocket, and attacked her before she realised his murderous intention and could offer any resistance. From the localised distribution of the blood marks, she evidently was unable to make any further movement; and he must also, without moving from the bed, there and then have cut his own throat, and fallen across her body in the position described.

The state of mind of the man, who harboured such an awful design for some time prior even to the arrival of his paramour in Edinburgh, as evidenced by his letters, and yet was able to dissimulate so perfectly as to spend an apparently happy time with her, and engage in the purchase of articles for their wedding, a few hours prior to the committal of the act, offers ground for interesting psychological study. I would merely draw attention to the fact that such behaviour is not inconsistent, in people who have shown no previous signs of mental aberration, with a pre-conceived and fixed design of murder, a design carried out in the present case with unrelenting and unswerving resolution against a person towards whom his professions and previous behaviour indicated a true, if not very deep, affection. The man's manner, prior to the deed,

gave no indication of the terrible scheme he harboured and foreshadowed in the letters written on the previous day, and this circumstance must be held to dispose of any theory of sudden homicidal impulse and subsequent suicide from grief at his act. There does not appear to have been any motive for the crime; there is no suggestion of unrequited love in their relationship, or of jealousy or infidelity, while to neither can be laid the charge of intemperance, one of the most common causes of crimes of this nature.

In fact, there does not seem to have been any other motive than simply that his circumstances in life did not enable him to fulfil his promise of marriage at the time he had fixed, and he therefore determined to end his life, and at the same time compel his betrothed to share his fate.

Meeting V.—February 7th, 1900

DR JAMES DUNSMURE, *Vice-President, in the Chair*

I. EXHIBITION OF PATIENTS

1. *Dr Alexander Bruce* exhibited a case of DISSEMINATED SCLEROSIS, in which a well-marked adductor clonus could be elicited when the patient was seated on a chair with the tips of his toes resting on the ground.

2. *Mr C. W. Cathcart* exhibited a patient from whom he had removed the cœcum and adjacent portions of the ileum and ascending colon for tubercular ulceration and contraction at the ileo-cœcal valve. The lad R. D., æt. 15, dated his illness from an attack of abdominal pain in May 1899. The pain was intermittent at first, but gradually grew worse, and he had to stop work. For nine weeks he was treated in the hospital at Stirling, and left it much improved, although not quite well. He remained pretty well until the beginning of October, when the pains returned, and he was re-admitted to the Stirling hospital. After eight weeks there he was sent to Ward 31 in the Royal Infirmary, Edinburgh, to be under Dr James, and a

few weeks later was transferred to Ward 5. During the greater part of his illness he had been troubled with diarrhœa. When admitted to the surgical ward he was much emaciated, and could only take a very limited diet, chiefly of milk, without much aggravation of his symptom. He had an almost constant diarrhœa and frequent attacks of abdominal pain. The abdomen was distended, and during attacks of pain the "ladder" pattern, indicating chronic intestinal obstruction, was clearly visible. The distended coils of bowel began at the right iliac fossa. On palpation a considerable thickening was recognisable in the right flank. From its position, and from the large size of the distended bowel next it, the obstruction was thought to be in the ascending colon. As the patient was growing steadily worse in spite of palliative measures, it was decided to open the abdomen in the hope that a condition might be found amenable to operation. On December 23rd, 1899, the abdomen was opened in the right flank. The disease was found to be in the cœcum, which was thickened all round and firmly adherent to the posterior abdominal wall. After some trouble the diseased part was freed and brought out at the wound, and after having been clamped above and below, was excised. The lower end of the small intestine resembled the large intestine, and was in fact larger and thicker than the ascending colon beyond the obstruction. The cut edges of bowel were united end to end with a double row of continuous silk stitches. For the first eighteen hours the patient's progress was fair, after that his heart began to flag, and the pulse went up to 160 per minute. Accordingly next day about 24 oz. of ¾ per cent. saline solution were infused into a vein. From this time he improved—slowly at first, then more rapidly. The wound has healed without any tendency to hernia, the diarrhœa has been replaced by regular daily action of the bowels with formed motions, and he has gained 16 lbs. in weight. He takes his food without restriction, and is free from pain.

The specimen (shown) is one of tubercular ulceration and contraction in the neighbourhood of the ileo-cœcal valve. With difficulty a rod about the size of a No. 6 catheter has been passed through the stricture, but as it was lined by soft granulations, its calibre would probably have been less during life. The diarrhœa was probably kept up by the congestion induced by the constant efforts of the bowel to overcome the obstruc-

tion, and this diarrhœa was no doubt most beneficial. Had the fœcal matter become solid or nearly so above the stricture, obstruction would at once have become complete. At the operation several enlarged and probably caseous glands were felt at a little distance from the part excised, but no attempt was made to remove them.

3. *Dr Norman Walker* exhibited a case for diagnosis. Patient was a girl, aged 20, with an ulcerated area the size of a double florin on the extensor aspect of the forearm. This had been present for a year, during which time it had once partially dried up. The diagnosis seemed to lie between syphilis and tuberculosis, and taking into account the chronicity of the lesion and the absence of any evidence of syphilis (scars, etc.), he was inclined to the latter view, in spite of the fact that there had been a little improvement while the patient took iodide of potash.

The majority of the surgical opinion, informally expressed, was in accord with this.

II. Exhibition of Specimens

1. *Mr F. M. Caird* exhibited (*a*) TWO SMALL SEQUESTRA the size of hazel-nuts removed from the alveolar border and hard palate of the upper jaw. They were both obtained from patients who had undergone a mercurial course during the primary and secondary stages of the disease. The first, L. W., an abstainer, infected eleven years ago in India, was treated for nine months by pill. Seven years ago destructive throat symptoms appeared—loss of soft and greater part of hard palate and of upper lip, with extensive pharyngeal ulceration and separation of sequestrum exhibited.

The second, M. L. W., infected eight years ago, was treated by the mouth and by inunction on the Continent. There were no secondary symptoms till later, and then only in the mouth.

(*b*) A specimen of EXTENSIVE SYPHILITIC NECROSIS involving the greater part of the frontal bone and both parietal bones. The patient, aged 20, suffered from hereditary syphilis, and showed characteristic manifestations of the disease. For many years she had sinuses discharging fœtid pus from the sagittal, coronal, lambdoid, and temporal sutures.

The preparation was removed after death and disclosed a huge cavity in which the surface of the brain, covered with its thickened dura mater, lay at the upper level of the squamous portion of the temporal bone. Various futile attempts had been made during life to perforate or remove the sequestra.

2. *Mr D. Wallace* exhibited a RADIUS (right) with old-standing fracture 1½ inches from carpal end. The displacement of lower fragments backwards was so extreme that the carpal surface was turned almost to the dorsal aspect.

III. Exhibition of Instrument

Mr J. M. Cotterill exhibited a new form of TREPHINE. The instrument had been modified and simplified from the varieties already in use, so that it now consisted of only two parts, without any screws or corners to harbour dirt; and Mr Cotterill claimed that the instrument, while working effectively, had the merit of extreme simplicity, while it could be very easily and very thoroughly rendered aseptic.

IV. Demonstration

Mr C. W. Cathcart explained the advantage of having gauze dressings rolled in the form of balls, which pull out from the inside.

If a piece of gauze, say 6 yards in length, is folded across its breadth till it becomes like a loose rope, it can be wound on a thick rod (see *Scottish Medical and Surgical Journal*, vol. xvi. page 177). It can then be placed in a box of the proper size with its inside end left projecting from a packing of cotton wool. The gauze can be drawn out and the required quantities cut off with scissors without disturbing or exposing the remainder.

The ball and its box can be sterilised apart from one another. Afterwards, with purified hands, the ball can be placed in the box and packed round with wool.

Messrs J. F. M'Farlan & Co. will issue, when requested, any of their medicated gauze dressings rolled in this way—such as iodoform, bicyanide, or boric gauze.

V. Original Communications

1. NOTE ON A CASE OF RUPTURED FEMORAL ANEURISM FOR WHICH THE COMMON FEMORAL ARTERY WAS TIED

By F. M. Caird, F.R.C.S.Ed., Assistant Surgeon,
Royal Infirmary, Edinburgh

The following case seems worthy of record on account of its comparative rarity. For the notes of the case I am indebted to Dr Veitch of this city, who called upon me to operate.

The patient was a strongly built and tall working man, whose staple diet was tea and bread. He was engaged in a rubber factory and had enjoyed good health till lately, when he had been troubled with chronic bronchitis. In the spring of 1899, however, he called upon Dr Veitch, complaining of two swellings which had appeared towards the inner aspect of each thigh. The swellings were of the size of a small tomato, and from their distensile pulsations and other characteristics were readily diagnosed as aneurisms of each femoral artery in Hunter's canal. No cause could be ascertained, and the patient stated that he had first noticed them after an attack of cramp.

He was advised to cease work, to rest, and to take Iodide of Potash internally; and this treatment was pursued till the end of July, during which period the condition remained stationary with the exception of increased pain in the right aneurism. The right aneurism now began to increase so rapidly in size that rupture was feared, but the patient emphatically rejected all attempts to get him into hospital. On August 21st, it appeared that the aneurism had given way, and on the following day this belief was confirmed. Extravasations of blood then extended from the buttock to the knee, pulsation could not be detected in the tibial arteries. The patient's sufferings were now very great, and compelled him, 23rd August, to face surgical relief. He still refused to be taken into hospital, but agreed to operation in the single room which he occupied and which served him for kitchen, parlour, bedroom and hall.

The limb from the knee down was found cold, numb and

pulseless. The condition appeared desperate, and it was decided to interfere at once. With lack of assistants, amidst foul surroundings, and in a small room, one was driven to select the most simple procedure, and accordingly the common femoral was exposed and ligatured with a stout piece of old carbolised catgut below Poupart's ligament. The limb was then enveloped in corrosine wool and the patient put back to bed.

The patient now became extremely intractable and restless. A Jubilee nurse did what she could during occasional visits to look after him, but he was out of bed twenty times a night; he had retention of urine, and hypostatic congestion of the lungs with bronchitis aggravated matters, his temperature running up to 103°. The dressings became disarranged and soaked with urine and had to be removed on the fifth day, when the wound was found healed by first intention, and in spite of complications, the patient gradually improved and was practically well on the 14th September. For some weeks his sole complaint was pains of the limb.

Towards the middle of November the patient drew attention to pains in the lower part of the hæmatoma. Signs of suppuration were detected, and on November 19th a quantity of slightly purulent fluid blood was evacuated by incision. The original aneurismal sac could be felt above the site of the incised swelling.

The patient is now well. The limb is somewhat weaker than its fellow. The aneurism (March) on the right side is so small that it is barely visible, hard and nonpulsatile, while that of the left side remains very much *in statu quo.*

In regard to the application of the ligature to the common femoral, it was felt at the time of the operation that the life of the limb hung in the balance; and while one saw that, under the circumstances, to ligate the external iliac in the person of a stout man with a somewhat large abdomen would be no easy matter, yet there was sufficient encouragement in the results gained by the Dublin School to warrant the practice that ended so happily.

The photograph taken in February shows the large mass formed by the aneurism and hæmatoma on the right leg, the scar of the incision at the base of Scarpa's triangle, as also the fellow aneurism, which remains stationary, of the right femoral. There is also left inguinal hernia.

2. ON THE INCREASE OF DISEASES OF THE NERVOUS SYSTEM AND OF INSANITY

By WILLIAM W. IRELAND, M.D. Edin.

AMONGST the physicians who have treated of the increasing nervousness of our time, none has been more prominent than Professor Erb, of Heidelberg. The last published discourse of this eminent neurologist on this topic may be read in the *Revue Scientifique*, 2nd April 1898. But if we are to overtake other parts of the subject, we cannot do more at present than to indicate the main points in Dr Erb's thesis. He dwells upon the increase of the causes of nervous irritation and depression, the greater anxiety, hurry, and mental toil of life, the overtasking at school, and the increased amount of irksome study men have to go through in order to enter the learned professions, the more exciting nature of their amusements, the more stimulating character of their diet, and the restlessness introduced into their lives through the facilities of inter-communication. The causes being thus increased, the effects are bound to follow. For my part I should have liked if the learned professor had essayed to pursue his deductions a little further, and afforded some direct evidence of this increasing nervousness, which, he says, has been going on for the last thirty years. Most of us are aware of the difficulty of earning a living wage at the outset, and of keeping up against unceasing competition, and of the greater excitement and complexity of life. It may be said that this strain falls most severely upon the middle classes. The diffusion of education amongst all ranks, the struggles of aspiring persons amongst the poorer classes to rise in the world, the discontent and bitter wars of labour, entailing incessant disputes and frequent strikes and breaches of contract, the spread of the co-operative movement which is rendering the position of the private shopkeeper more difficult to hold, and the formation of big companies which crush out small businesses; all these, and some social antagonisms on which we have no time to dwell, go to make it a harder and less agreeable world to many.

At the same time the new conditions of our to-day life are not without their compensations. If we move about more, we

obtain more frequent amusement and change of air; if letters and telegrams too often claim our attention and disturb our repose, they often banish uncertainty and anxiety. Sounder views on dietetics have become known, and drunkenness has become less prevalent; the decrease in intemperance in liquor amongst the middle and upper classes during the last half century has been very marked, and there is reason to believe that this decrease is extending to the lower classes. In general we lead more secure lives than our fathers did, live under better laws, and have less to fear from injustice and oppression. There can be no doubt that during the last half century the condition of the working classes has greatly improved, their wages are doubled, while the purchasing power of money has increased for most articles, and they live under much better sanitary conditions. The result has been a notable diminution of the death-rate both in England and in Scotland during the last quarter of the century. Nevertheless it appears to me that the strain upon the nervous system is getting greater, at least for the middle and upper classes, and that therefore we may expect more nervous derangement and breakdowns. But to make a general opinion like this evident by exact inquiries is, on the face of it, very difficult.

There are some statistics to answer the questions: Is insanity increasing? Is idiocy increasing? And is the number of suicides increasing? But the inquiry whether other forms of diseases of the nervous system have become more frequent can scarcely be approached in that way, as many such disorders do not affect the mortality returns or appear as results in statistical tables of any kind. Anxious to obtain the opinions of physicians of experience and reputation on this question, I addressed to a number of medical men the following queries:

1. Have you found since you began to practise medicine that there has been an increase in the relative frequency of diseases of the nervous system?

2. Have new forms come into prominence?

3. Have you observed any change in the type of old diseases?

To these circulars I received a good many interesting answers, some of them from men whom I only knew by reputation. Several of my correspondents took the occasion to point out the dangers of fallacy in answering these ques-

tions. Medical men generally begin to practise amongst the poor, and in the course of time come to have more patients amongst the richer classes, who are subject to diseases of a different character. Some physicians gain in the estimation of the public a reputation for the treatment of specific maladies or diseases of different organs, and thus gain more patients of one kind than another. The most trustworthy answers towards our inquiry might be expected from medical men who had long practised in one district in the country, or in small towns, treating every class of patients alike; but these gentlemen who have done me the favour of answering my inquiries all declare that they see no reason to affirm that diseases of the nervous system have increased in their districts relatively to other diseases. Some physicians of experience practising in large towns are of the same opinion. Sir W. T. Gairdner and Dr MacVail in Glasgow, Dr George Balfour in Edinburgh, Dr Leech in Manchester, Dr David W. Finlay in Aberdeen, Dr Henry Barnes in Carlisle, and Dr Grieve, late Surgeon-General of British Guiana, have noticed no increase in the relative frequency of nervous diseases.

We may spare time for the reply of Dr A. M. M'Aldowie, of Stoke-on-Trent: "I should be inclined to say off-hand that nervous diseases are more frequent now than formerly; but on examining the facts, I feel convinced that the increase is apparent only, and is due to the fact that one recognises early system lesions, neurosthenia, etc., in much slighter forms than fifteen or twenty years ago. On comparing my case books (I keep a record of every case I see, in private or hospital), I cannot find that diseases which were well recognised at the period I mention, *e.g.* epilepsy, chorea, locomotor-ataxy, are more frequent now than then. My own opinion, therefore, is that diseases of the nervous system are not on the increase."

Some physicians point out that there has been during the past twenty years a greater differentiation in the diagnosis of nervous disease, and that the nervous element in ordinary diseases is better recognised than it used to be.

On the other hand, some physicians of reputation in the same towns, but presumably treating a different class of patients, have stated their opinion that nervous diseases have increased in frequency during the last twenty years.

Some of the medical men in their replies nicely balance

this and that consideration, and hesitate to pronounce a formal opinion. The only physician, save one, in the United States to whom I have written—Dr Osler, of Baltimore—is of this neutral class.

Amongst those who have given a categorical reply that they believe nervous diseases are on the increase, are Sir Frederic Bateman, of Norwich; Dr Eddison, Leeds; Dr James Goodhart, London; and Dr Ernest Reynolds, Manchester. Dr A. James has taken some trouble to further the inquiry, as may be seen from his letter: "I have been looking up the ward journals of the Infirmary, and I find in the late Professor Sanders' journals, 1869-1878, there were 144 nervous cases out of a total of 1519 male cases. In my own journals, 1892-1899, there were 155 nervous cases out of a total of 809 male cases. This gives a tremendous increase in the nervous cases. I thought there would be an increase, but I did not expect such a great one. Surely there must be some fallacy, but I cannot yet see where it comes in. I chose Sanders' journals because I knew them best, as I was a clinical clerk, and resident with him."

I also venture to add part of a letter from Dr Julius Althaus:

"1. I consider that the relative frequency of diseases of the nervous system has decidedly increased. This increase, however, holds only good for functional or nutritional diseases, and not for organic affections of the brain and spinal cord. Of these latter, those which are acknowledged to be owing to syphilis, have, in my experience, decidedly diminished during the last twenty years.

"2. The functional diseases are now prominent over structional ones, but I cannot say that actually new forms have sprung up in our time, except perhaps the neuroses owing to influenza. Of these latter no, or next to no, mention is made in the older writings, but of course this does not prove that they have not occurred. It appears to me more likely that the old physicians have failed to see the connection between influenza as an exciting agent, and the nervous sequels of it. This would be analogous to the influence of syphilis. Hunter, and even Romberg, denied that syphilis ever caused lesions of the nervous centres, but it seems impossible to assume that in their time syphilis did not act in the same manner

as it does now, in producing an immense variety of nervous affections."

As Dr C. H. Hughes, of St Louis, U.S.A., Dr David Drummond, of Newcastle, and Dr Urquhart, of Murray's Royal Asylum, Perth, like Dr Althaus, are well known as neurologists, their opinion that nervous diseases are much increasing may be held merely to signify that such patients are likely to drift to them. Dr George Keith is decidedly of opinion that nervous diseases are much increased since he began to treat disease almost sixty years ago. He attributes it in great part to the abundance of flesh meats now allowed to children. Dr Strachan lays much stress upon the evil effects of overtasking children at school, a subject to which he has given much attention. Most physicians who believe nervous diseases to have become more frequent, consider that the increase falls in the lighter forms, especially in neurasthenia.

Few physicians have noticed any change in the type of diseases. Dr Leech, who has noted none in nervous complaints, has observed a distinct difference between the rheumatic fever seen in earlier times than that seen now.

Dr Byrom Bramwell is inclined to think that there has been an increase in some of the functional neuroses, and he has definitely formed the opinions that disseminated sclerosis has, during the past few years, become commoner, and that this increased frequency is probably due to the fact that in many cases this sclerosis follows, or at least seems to have its starting point in influenza. He also thinks that the grandiose ideas in general paralysis are now often wanting. But this, he thinks, may be because those cases in which such exalted ideas are not observed were not formerly classed under the heading of general paralysis.

Dr William M'Lachlan of Dumbarton has no doubt that diseases of the nervous system are increasing. He has observed an atonic irritative condition to be more common. The patients feel that they are on the verge of losing their mental balance. The causes of this are commercial competition, social aping at what the income must be strained to meet, stock exchange gambling to catch fortune by a few lucky deals. In the female, sexual indulgence and the using of measures to obviate pregnancy—uterine disappointment followed by motor and volitional disturbance. Acute sthenic diseases are not so

common. Pneumonia and fevers are more of the adynamic type than when he first met them twenty-five years ago. Hysteria is not so common; tennis and the cycle are helping to give it a quietus.

This recalls the old question of a change of type in fevers and inflammations which was debated half a century ago between Doctors Allison and Christison on the one side, and Dr Hughes Bennett on the other.

Dr Halliday Douglas, long physician to the General Post-Office at Edinburgh, saw many cases of nervous derangements and failures of health amongst the employés, especially those who did their work under high pressure. The female clerks often required leave of absence from illness.

I have learned from several sources that monotonous work demanding severe and unremitting attention is most trying for the nerves and assists in causing mental derangement. Thus nervous diseases and insanity are especially common with telegraphists, letter-sorters, and persons who have to check mechanical labour.

From all the answers which I have received I should judge that there has been no noticeable increase in the number of diseases in the country districts and in small towns, but there is some evidence that there has been an increase in those complaints in large towns, especially in functional disorders such as neurasthenia. Probably this increase affects some classes, and persons of special pursuits more than others.

Graver Forms of Nervous Diseases

A physician interested in my inquiries who held that the increase was mostly in the lighter forms of nervous diseases was inclined to think that there might be fewer cases of the graver forms; but a study of the registers of mortality showed me that the contrary was the case. I spent several days, through the courtesy of Dr Blair Cunynghame, in the Register Office examining the statistics there. One thing appeared at the outset, that those diseases of the nervous system which are assigned as causes of death are less frequent in Scotland than in England. To quote the Registrar's report: While in Scotland in 1857 only 170 died of Brain and Nervous diseases in every hundred thousand, in England during the same year 273 deaths occurred in a like population from the same causes,

and year after year the relative proportions remain somewhat the same in the two countries.

In Scotland the proportion of deaths from Diseases of the Brain and Nervous System for every hundred thousand of the population was—

	Total for Scotland	Males	Females	In Towns			Mainland Rural	Insular Districts
1855	168	191	148	213			148	97
1856	161	179	146	203			143	81
1857	170	192	151	223			144	94
1858	169	192	148	211			148	104
Average of ten years from 1855 to 1864	170 both sexes			214			147	97
1865	179	199	161	233			149	113
				Principal Towns*	Large Towns	Small Towns		
1875 †	217	239	198	245	285	208	185	96
1885 †	231	248	214	264	252	226	209	95
1895	226	233	219	237	249	224	206	125
1896	206	211	201	215	221	206	190	144
1897	221	233	209	234	231	220	199	173

The general death-rate for Scotland was in 1855, 20·6 in the thousand; in 1897 it was 18·8 in the thousand. That is, to come closer, in Scotland there died of brain and nervous diseases in 1855, 168 persons to every hundred thousand of the population; in 1865, 179 persons; in 1875, 217 persons; 1885, 231 persons; and in 1897, 221 persons. The increase from 1855 thus amounting to 53 persons, about one-third of the first number. The proportion of deaths from different diseases in every ten thousand persons from diseases of the nervous system was—in 1855, 818; in 1875, 930; in 1885, 1210; and in 1897, 1109. In the report for 1897 we are told that from diseases of the brain and nervous system there were 9257 deaths, giving a mortality rate for Scotland of 221 in every hundred thousand, and forming 11·79 per cent. of the total deaths. The rate for Scotland is exceeded by the principal and large town districts; the lowest fall in the insular rural districts. Among principal towns the highest rate falls in Perth, 330; in Paisley, 288; in Coatbridge, 262; in

* This is a new sub-division of the Registrar.

† At Table XXVIII., of Report XXVIII.

Greenock, 250; in Glasgow, 238; in Edinburgh and in Aberdeen, 232. The nervous diseases specified are apoplexy, paralysis, epilepsy, and convulsions.

One can only guess at the efficacy of the causes of nervous disease in the gross. Drunkenness should be a potent cause; but there has been noted a decrease in the number of deaths from delirium tremens and chronic alcoholism in the last twenty-five years. There was also an increase in deaths from diseases of the urinary organs,[1] which were doubled from 1855 to 1897. Diseases of the circulation had also doubled during the same period. The mortality from phthisis had fallen from 261 in 1855 to 172 in 1897.

I did not make the same careful examination of the vital statistics of England, as this has already been done in the book of Dr Newholme on that subject, published last year. He shows that while there is a great improvement in public health as indicated by a lower death-rate; commencing in 1872 it fell from 22·6 in the thousand down to 17·4 in 1897. Dr Newholme says that "it is doubtful if the increased strain of modern life exists in the community as a whole. Assuming, however, that over-pressure exists in certain stations of life, *e.g.* among city merchants, medical men, etc., it cannot be said generally to exist among professional men; clergymen, lawyers, and civil servants are as classes long lived." But clergymen and civil servants generally lead easy lives, and it is likely that anxiety and over-pressure may be felt less by the lawyers than by their clients. "Even assuming," goes on Dr Newholme, "that over-pressure exists throughout the whole of the professional classes, these do not form the mass of the community. The majority of the population of England and Wales belong to the wage-earning classes, and the condition of these classes will therefore necessarily have the greatest influence on the total result." It is easy to prove that the working classes are now in a more prosperous condition than they were half a century ago. Newholme, however, shows that there is a high death-rate from diseases of the nervous system amongst barristers and solicitors, railway stokers, and medical prac-

[1] Urinary diseases, which comprise Bright's disease, acute nephritis, calculus, and affections of the bladder and prostate, stood in 1855 at 24, and in 1897 at 49 to the hundred thousand. During the same time circulatory diseases had risen from 78 to 170.

titioners. Diabetes, a disease connected with disorders of the nervous system, has steadily increased, as also renal diseases, which are high amongst doctors, lawyers, and innkeepers. Diseases of the nervous system show a death-rate per million of 1546 in 1861-65, and of 1600 in 1891-5. He observes: "In 1861-65 the mean death-rate registered as caused by intemperance was 42 per million. It steadily increased in 1891-96, reaching 68 per million. This increase is almost certainly due to more correct certification of deaths." At the same time the death-rate ascribed to cirrhosis, which I take to be generally the result of intemperance in drink, has increased amongst males from 43 in 1861-70 to 140 in 1881-90. There is also an increase in the death-rates from premature births and congenital defects.

Dr S. A. K. Strahan, in his book upon "Suicide and Insanity" (London, 1893), calls attention to the fact that, "while the general death-rate for England and Wales has fallen 16·4 per cent. during the past quarter of a century, a rise, in some cases amounting to over 100 per cent., has taken place in the death-rates from hereditary and degenerate diseases."

From a table compiled by Dr Strahan, it appears that the death-rate in England and Wales in the twenty-five years between 1866 and 1890 had increased for nervous diseases by 10·2 per cent.; from diabetes, 103; from kidney disease, 63·4; and from heart disease, 65·9.

From these statistics it seems to me that it is not going in advance of the evidence to pronounce that the graver forms of diseases of the nervous system have increased in Great Britain, as they are said to have done in France and Germany, during the last forty-five years. In Scotland there seems to have been a stoppage in the increase from 1885 to 1897, which, I hope, indicates that the rise of the death-rate from these maladies is not fatally progressive.

Suicide

Since the publication in 1879 of Morselli's work, "Il Suicidio," the attention of sociologists has been directed to the portentous increase of suicide in Europe and North America. This increase is most marked in great cities, and

seems to be steadily progressing in every country in Europe save Norway. It is needless here to repeat what I have written in my paper on "The Causes of the Increase of Suicide" in the *Journal of Medical Science* for October last.

On this subject Dr Strahan observes: "Taking the recorded numbers as being relatively accurate, we find that in the twenty-two years, 1867-88, the number of suicides has risen steadily. In the former year there were 1316 discovered suicides in England and Wales, and in the latter year 2308, which gives an increase of over 75 per cent. in the twenty-two years. That increase of population is not responsible for this difference is shown by the fact that, while the rate was only 61 to the million persons living in 1867, it had attained 82 to the million in 1888, an increase of more than a third within the twenty-two years.

"This steady and rapid increase of self-destruction is common to the whole civilised world. It is most marked in those countries which take leading parts in the world's doings, but it is noticeable in all. Dr D. R. Dewey has recently been studying the question in the New England States of America. He finds that since 1860 suicide has increased about 35 per cent. In Massachusetts it has increased in thirty years (1860-90) from 69·9 to 90·9 to the million living, and in Connecticut from 60·6 to 103·3 per million."[1]

In Scotland the rise from 1865 to the quinquennial period of 1890-94 has been stated to be from a rate of 40 to a rate of 54, an increase of 35 per cent. From my own inquiries I have found that in 1881 there were 182 suicides in Scotland—131 males and 51 females—out of a population of 3,735,573, giving a proportion of 48·7 to the million. During the quinquennial period within the years 1881-85 the mean annual number of suicides was 202, equal to 52·05 to the million. In the quinquennium, 1886-90, the mean number of suicides was 227, giving 56·4 per million. In the quinquennium, 1891-96, the mean number of suicides was 247, giving a proportion of 59·2 to the million. In the year 1896 there were 297 suicides—216 males and 81 females. This gives a proportion of 71·2 to the million.

As the causes which tend to produce suicide are of the

[1] "Suicide and Insanity," p. 186.

same character as those which tend to produce insanity, greater sensibility and irritability, and a lessened power of reaction, it is not surprising that those who have committed themselves by denying that insanity is increasing should be ready to question any rise in the number of suicides. Their position is incongruous, for as they advance that the increase in the number of registered lunatics is owing to the increased number of admissions of early and milder cases of mental derangement, they have to escape the awkward question: Why, since many of these cases are of a pronounced suicidal character, is there not a decrease instead of an increase in the gross number of suicides? As the statistics in both questions are against them, they have to show that the statistics which indicate an increase of suicides are "crude," and then to show that the uncooked statistics which reveal an increase of lunatics should be looked upon with disfavour. Apparently they will be content with a verdict of "not proven."

It seemed to me that enough had been said about Dr Sibbald's doubts of the increase of suicides in Britain in considering "The Causes of the Increase of Suicide" in the *Journal of Medical Science* and in the debate thereon; yet since Dr Clouston gave his opinion that I should not have passed this by, and Sir John Sibbald has repeated his views before a learned society, of which I am not a member, I venture to add a few words. He lays great stress upon the fact that suicides by hanging have not increased in England and Scotland during the last thirty years, while self-destruction by other methods is reported as increasing. There seems no reason for presuming that the means used for suicide should always keep the same proportion. It is known that in the fashion of making away with themselves men are much influenced by imitation, and during the last thirty years hangings have been much rarer and no longer a public spectacle. Practically death by strangulation has been displaced by dislocation of the spinal cord. With the spread of information easier and less repulsive methods of death are likely to be selected. Less conservative people, like the native-born Americans, have mostly given up hanging for self-destruction, hence we need not be perplexed that it in these islands lags behind the general increase of suicides.[1]

[1] I have found that in Scotland during the five years between 1881-85 the mean

There has been no change of any consequence in the manner of inquiring into sudden deaths in Scotland. Self-destruction is still regarded as a deplorable event in the family; but there has been no opposition to burial in the churchyard for many long years. While some suicides are put down as being drowned or being killed by accident, few accidental deaths are registered as suicides. Altogether, though there are no doubt stray errors and omissions in the registers, these are not sufficient to vitiate the general results.

Idiocy

The statistics of idiocy taken from the census are untrustworthy, owing to the reluctance of parents to admit that their children are so affected. Wildermuth has observed that the number of idiots is decreasing in Würtemberg; but we think this only indicates a diminution in the cases of endemic cretinism, which is everywhere declining in number owing mainly to the people being taught to avoid drinking water from sources which are known to cause goitre and cretinism. In my book "On Idiocy and Imbecility," published twenty-three years ago, statistics were given showing that there had been a fall in the number of idiots in Norway between 1855 and 1865 of 14·63 per cent. Unfortunately this has not been sustained. In 1865 there was one idiot in every 834, and one insane in every 524 inhabitants; but by the census of 1876 there was one idiot in every 498, and one insane person in every 398. It was hoped at first that this unpleasant result might be owing to the questions being put in so indefinite a manner that mistakes were made in the answers. In the census of 1891, which was carefully taken, there was one idiot for every 823 inhabitants, and one insane person for every 376. In Sweden there was found to be, in 1860, one insane person to every 771 of the population; in 1890 there was one lunatic to every 550. In 1860 there was one idiot to every 1542 persons, in 1890 one to every 628. These statistics thus bring

yearly number of suicides by hanging was 57, giving a proportion of 14·6 to the million. In the quinquennium of 1886-1890 the mean number of such deaths by hanging was 66·8, equal to 16·6 to the million. In the quinquennium, 1891-1896, the mean number of suicides by hanging was 66·8, giving a proportion of 16·01 to the million. Thus even suicides by hanging had increased during the last two quinquennial periods over the first. In the year 1896, out of 297 suicides, 69 were by hanging, giving a proportion of 16·5 to the million.

out an increase both in idiocy and insanity in Sweden and in Norway, although we have been assured that drunkenness has much diminished in these two countries owing to the working of the Gothenburg system.

By the last census the number of idiots in Ireland was stated to have fallen from 8639 in 1881 to 6243; in 1881 there was one idiot to every 598 of the population; in 1891 one to every 754. The number of lunatics had increased from 9774 to 14,945.

Increase of Insanity in England

The steady and progressive increase of the number of the insane throughout Europe has now begun to attract public attention. Much of this increase is due to collection of lunatics in special asylums, and to their accumulation, owing to better care giving some of them a longer life; but this fails to explain the whole rise, and physicians like Koch, Kollmann, Cettingen, Lunier, and Kraepelin, who have devoted time and care to examine the statistics have, after making every allowance, come to the conclusion that there has been a real increase in the absolute number of the insane. Unhappily the Commissioners in Lunacy for England took a different view, and as the number of lunatics they had to deal with perversely kept on increasing, they have had much care to explain away their own statistics. There were always, however, some men well acquainted with the subject to show that there was a real increase, Sir James Crichton Browne amongst them, and the phenomenal rises in the number of "official lunatics" in 1897 and 1898 have opened many eyes. In 1897 there was an increase of 2919 registered lunatics in England and Wales, and in the next year an increase of no fewer than 3114, the largest annual rise which the Commissioners have had to record. In 1859 there was one official lunatic to every 536 persons. In 1869 there was one to every 418. In 1879 one to every 363. In 1889 there was one registered lunatic to every 337 persons, and in 1898 it was one in 302. For the last ten years there has been a yearly increase of over 2000 lunatics.

The inference that this denotes a real increase is confirmed by the returns of the last census. In 1871 there was stated

to be one insane person to every 329 of the population; in 1881 there was one insane to every 307, and in 1891 one to every 298.

The following is taken from the *Journal of Mental Science*, April 1899. "Dr Bowes has issued a most interesting report on the existing high rate of lunacy in Yorkshire. The first question discussed is whether the apparent increase of insanity in England is real or only due to accumulation of the chronic insane and the removal of many quiet dements from their homes. Dr Bowes agrees with the general impression that there is some increase, but not enough to create alarm. He points out that there is a much larger proportion of insane to sane among the agricultural labourers than among miners and city dwellers. He shows that for thirty years the agricultural counties have shown most pauperism and most insanity. He thinks that marrying in and the poorness of the living and generally depressing vital conditions account for this. All this is true, but we believe that it is true also that the more feeble are left in the country to breed degenerates and to live from hand to mouth."

A considerable increase in pauper lunatics has also been noticed in the counties of Argyleshire and Inverness-shire, where all the more energetic inhabitants betake themselves to other places for employment. Nevertheless there may be exciting causes of insanity acting even in the rural districts in this unquiet age. We have seen that the graver forms of nervous diseases are increasing in the insular districts of Scotland.

Dr Hastrup, a Danish physician,[1] has noted that new nervous diseases have appeared even amongst the Esquimaux, which some believe to be owing to the great use of coffee, which the Esquimaux obtain from the traders in exchange for skins.

Insanity in Scotland

The same steady increase in the number of the insane goes on in Scotland as in England, and there has been the same startling rise in 1897 and in 1898 which has distanced all arithmetical explanations. In 1898 there was an increase of registered lunatics of 487, of whom 49 were private patients

[1] Hospitals Tidende of Copenhagen for August 11 and 18, 1886.

and 438 were pauper patients. At the beginning of the present year the figures furnished by the General Board of Lunacy give since 1858 up to 1898 a net increase of 9575 in the number of lunatics under the jurisdiction of the Scottish Lunacy Board, or 64 per cent. The increase of the population has been only 41 per cent. The census returns for Scotland showed in 1871 one insane person for every 494 of the population; in 1881 one insane for every 444; and in 1891 one insane for every 385.

The author of an able communication which appeared in the *Scotsman* on the "Growth of Insanity in Scotland," who has evidently great command of information, writes that the average increase may be put down as at 2·4 per cent. yearly, or more than double that of the population increment; but where the position is adjusted by deducting the deaths, removals, and recoveries, averaging 1·2 per cent. per annum, there is still this balance of over 1 per cent. to explain the accumulating process, which accounts for the swelling totals. The author of this communication further observes: "There is not much to justify the expectation of a higher recovery rate under present conditions. For quinquennial ending 1879 it stood at 16·5 per cent. on the average number on the register; in 1884 it fell to 15·5; in 1889 to 13·3; and in 1894 to 13·4."

The death-rate is much the same as it was in 1858. In some asylums which have many admissions from a city population it is even higher. Dr Clouston has remarked in his annual report for the year 1898 that the death-rate in Morningside within the first twelve months after admission has been steadily rising from 46·4 per cent. in 1874-78 up to its present rate of 51. It has been advanced by those who combat the belief that there is a real increase in insanity in Scotland that there is little increase in the number of private patients, and that the rise is mainly in pauper lunatics; but when one looks at the real naked facts he sees that it could scarcely be otherwise. During the last forty years the smaller asylums which took in those who could pay moderate boards have been squeezed out of existence. The few private asylums remaining are obliged to charge much higher, and the general expenses for the upkeep of the insane have much risen. For many years back it has been the custom that those bearing the

burden of a lunatic relation who are anxious to pay what they can, go to the inspectors of poor to gain admission into the district asylums. The patient is certified to be a pauper lunatic and his board in whole or in part is paid by the relatives to the inspector of poor, who transmits it to the treasurer of the asylum. Thus a considerable number of those who are what would formerly have been private patients are now officially returned as pauper lunatics, although they are not really so. This probably leads to looseness of administration, and no doubt makes it easier for people to get rid of the burden of supporting a lunatic dependent than formerly.

Insanity in Ireland

The general report of the Census Commissioners shows that the number of lunatics in Ireland returned in 1891 was nearly treble the number in 1851, and that the increase between 1881 and 1891 was from 9774 to 14,945, while there was a decrease of 9·1 per cent. in the general population. The population of Ireland in 1851 was 6,552,385; in 1881, 5,174,836; and in 1891, 4,704,750. From the Eighth Report of the Inspectors of Lunacy in Ireland, it appears that the total increase during the year 1898 was 714; it was larger than that for 1897, viz., 624, and exceeded the average increase for the past ten years, viz., 444. In the Report for 1897 the inspectors said that the rise was mainly the result of accumulation brought out by a low death-rate and a diminished number of discharges. They considered the number of admissions to asylums a more trustworthy source of information. In fact the admissions were less that year (1897), and in Ireland the admissions do not increase every year, but when we take the sum of every two years we find that they are progressively larger. The admissions rose in 1898 higher than they had ever been.

Of the causes assigned for this increase we are willing to allow something to the accumulation of chronic cases in asylums, the greater readiness to grant admission to all cases of mental failure, aided by the four shillings grant first given from the Imperial Exchequer twenty-five years ago to ease the local rates. Much stress is laid upon the greater willingness of the friends of lunatics to send them into asylums owing to

the increase of confidence and the skilful and humane way in which lunatic asylums are now managed. This, of course, is an argument most pleasing to the superintendents of these institutions. It is capable of indefinite extension. A sudden leap upwards in the admissions only means that the public confidence in asylum stock had risen. I am far from insinuating that this increased confidence or lessened distrust is not what it should be, but I am doubtful of its extent and intensity. It is but ten years ago since, owing to a popular outcry of suspicion, the superintendents in England were shackled with a new Lunacy Act, bristling with galling restrictions and insulting returns. Few people know anything about asylums till some of their relations go mad, and then they get them out of the house simply because they have become intolerable in it. What keeps them back from doing so is mostly the expense, which is assuredly not diminishing, for patients whose friends can pay. Moreover, the probability of heredity of insanity or idiocy is now getting diffused, and has begun to render people wary of making a damaging admission by sending their kindred into institutions which are becoming ever more public. While granting something to all these explanations, it is the outcome of my inquiries that they fail to account for the whole increase of insanity. Again and again have these purveyors of explanations been obliged to make their premises bigger to account for the swelling numbers of the last few years. They remind me of a man who has pronounced that the sea tide was not rising, and who persists in imagining that every falling back wave was bigger than the incoming one, while all the while he is again and again driven to change his position by the flowing tide which represents facts and not arguments.

There are men in the country who have the spirit to look at things as they are, and these I should ask to recognise that we have to deal with an advance along the whole line, an increase in nervous diseases, in suicides, and in lunacy.

After this paper was finished I took up the last edition of "Psychiatrie,"[1] by Dr Emil Kraepelin, where I found a complete agreement with my views. I translate one passage: "On the other hand, regular census with us indicates with certainty a

[1] "Psychiatrie ein Lehrbuch für Studirende und Aertzte." Sechste Auflage, Leipzig, 1899. 1 Band, p. 88.

rapid increase of the insane which far overtops the general increase of the population. No doubt this rise is partly accounted for by the greater care of the numeration and the better knowledge of insanity. Nevertheless, I believe we cannot any more doubt that we have actually to reckon with a very considerable increase of insanity. This is proved, not only by the alarming increase in the number of the insane, but also by the simultaneous rise in the frequency of suicides, and the contrast presented by the town and rural populations."

The learned professor observes that we are living in a time of transition, and he hopes that coming generations will be able to enter on the stress and struggle of life with fresh strength and better weapons. We need not believe that this increase of diseases of the nervous system is destined to fatal progression; it may stop or it may retrocede ere long. It would be rash to assume that we are acquainted with all the causes, and the means of prevention are not entirely beyond our control

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Meeting VI.—March 7th, 1900

MR A. GORDON MILLER, *President, in the Chair*

ORIGINAL COMMUNICATION

ON HEREDITY IN DISEASE

By D. J. HAMILTON, M.B., F.R.S.E., Professor of Pathology in the University of Aberdeen

ALLOW me, in the first place, to thank the Council of the Society for the very great honour they have conferred upon me in asking me to take part in this discussion, and more particularly for the place they have assigned to me in the order of debate. When I think of the many members of this Society who have made heredity a special study, I feel the honour to be all the more flattering.

It will be acknowledged on every hand that the subject you have chosen for debate is full of interest; an interest which has become, if anything, more intense in late years than formerly, from the vast amount of literature, biological

and purely medical, which has found its way into our midst, dealing not only with concrete examples, or supposed examples, of acquired characters being hereditarily transmitted, but going to the very root of the matter in speculations as to what heredity depends upon, and of what the mechanism consists which underlies its manifestations. It is one of the commonest experiences of life that not only the general conformation of the body may be transmitted from parents to child, but that the same holds good of the minutest details of conformation. Further, it is matter of common note that the most subtle mental features and traits of character may, similarly, be handed on from one generation to another. We have little experience of how far the finer manifestations of heredity prevail in unicellular organisms, but in the higher metazoa the phenomena are so blatant as to have attracted attention in all ages.

The subject of discussion this evening is the influence of heredity in disease, and here it will be advisable to recall the fact that mere congenital transmission of disease from either parent to child is not heredity. Most of those diseases which are transmitted to the fœtus *in utero* are of the contagious type, and, consequently, are to be referred back to the action of a microphyte. Such transmission of contagious disease is, of course, to be accounted for by direct inoculation of the fœtus, in most cases through the system of the mother. It used to be supposed that the placenta was a perfect filter against the transmission of contagion of the particulate kind; it was taught, for instance, that anthrax is never conveyed from the blood of the mother to that of the embryo. And although the approach of the placenta to perfection in this respect is undoubted, yet now and again it fails, and the contagion passes over to the offspring. Diseases of the contagious type seem to differ in the facility with which they are transmitted by this means. Thus, in the case of anthrax and tuberculosis, the infection of the fœtus through the mother occurs only very rarely, while we know that in that of syphilis the liability is extreme. These cases, however, of intra-uterine contagion hardly come within the scope of our discussion, unless in so far as they can be utilised for the purpose of proving or refuting the alleged transmission of a disease which has been acquired.

Of all the numerous theories to account for the handing down of hereditary characteristics, there may be said to be two which have met with most acceptance; not that they have much in common, but rather because they represent the two points of view from which the matter is regarded. Indeed, it may be said that nearly all other theories which have gained currency are to be looked upon as modifications or extensions of these. The one, it will be remembered, is known as the "Pangenic Theory" of Darwin, propounded by its author, with his characteristic modesty, as a mere provisional or tentative explanation of how acquired features may be transmitted; the other, that of Weissmann, known as the theory of the "Continuity of the Germ-plasm."

Between what we recognise as living protoplasm and its ultimate molecules, there are probably several stages of subdivision, molecular groupings, to the lowest of which various names have been given in keeping with certain theories of vitality bound up with their supposed existence. Thus these supposititious supra-molecular bodies have been termed pangens (de Vries), gemmules (Darwin), micellæ (Nägeli), idioblasts (Hertwig), plasomes (Wiesner), and biophores (Weissmann). They all refer to what may be regarded as molecular aggregations, which, by their association and interaction, go to make up what is roughly termed in biological language "living protoplasm." According to Darwin, these excessively minute particles of living matter, "gemmules," as he named them, are constantly being given off from the body-cells and circulate freely throughout the system. They were reputed by him to collect in the reproductive-cells, and to imprint upon these the features which the somatic cells had acquired through external agencies. Thus they tended to proceed centripetally, to convey to the reproductive-cells those impressions which had been received from without, and to transmit these to the offspring. The inheritance of acquired characters, according to Darwin's hypothesis, was therefore habitual.

Weissmann, on the other hand, takes the view that germ-cells (ova and spermatozoa) and somatic-cells differ, in so far, that the germ-cells are the exclusive bearers of hereditary qualities, the transmission being effected through the instrumentation of what he calls the "germ-plasm." This "germ-plasm" is comprised in the chromosomes of the ovum and

spermatozoön, is continuous from one individual to another, and is, therefore, practically immortal. He would regard the germ-cells as an exclusive royal line, as it were, handed on from generation to generation, and having no community, so far as the propagation of hereditary qualities is concerned, with the cells of the soma or perishable part of the organism. All new hereditary features are the result of variation in the structure of this "germ-plasm," and are brought about by the recurrence of slight inequalities of nutrition. He ignores the influence of external agencies acting upon the somatic-cells as an element in the production of heredity; he believes that only those variations which are blastogenetic become hereditary, those which are somatogenetic are not so. His theory supposes that the particulate elements of heredity proceed exclusively in a centrifugal direction, and is therefore opposed to that of Darwin. The inheritance of acquired characters, according to his theory, is impossible.

It will be evident that the whole matter of the alleged transmission of disease hereditarily hinges upon this debated point, as to whether acquired characteristics can be inherited; in other words, whether external agencies can so impress the soma, and through it the "germ-plasm," that the effects of these impressions become hereditary. Hæckel, Virchow, and Eimer are at one with Darwin in the belief that they can, and the evidence they have brought forward in support of the assertion is voluminous, in many cases striking, but by no means in all instances convincing. Virchow is a strong upholder of the theory of *causæ externæ* generating effects which are inherited, and it matters not whether these act on germ-cell or body-cell. "A living being placed under varying circumstances changes its functions and habits, and what it acquires it can transmit hereditarily." On the other hand, we have Weissmann, Ziegler, and others, just as strongly convinced that acquired properties are not inherited, but that hereditary tendencies are due to variation in the structure of the "germ-plasm."

The supposed transmission of *mutilations* I shall pass over in a single word by asserting that there is no evidence in support of the view. The Jewish and other Eastern nations have been circumcising since the days of Abraham, and pro-

bably long before that period, and I have yet to learn that Jewish children are born without foreskins.

Can we say, however, that an external agent of any kind giving rise to a morbid state of the body, ever so impresses the cells of the body that the disease becomes established as a hereditary peculiarity? Let me take up some of the best known examples of so-called hereditary diseases with the view of discussing them, and of calling forth the valuable experience which members of this Society must be able to bring to bear upon the subject.

Perhaps no disease is held to have so hereditary a tendency, at least in the layman's mind, as tuberculosis; and it is undeniable that the disease affects different members of the same family, and that the predisposition to it can be traced back in the ascendants as a distinct family strain. If I had come before this Society previous to the discovery of the tubercle bacillus, and had asserted that the disease is not hereditary and very seldom congenital, my remarks would have met with little credence. Yet such is the general conclusion of pathologists at the present day. With extremely few exceptions—so few that they may almost be neglected—children are not born tubercular even of tubercular mothers, nor are the young of the lower animals born tubercular under like conditions. Even in these exceptional cases there is the possibility that the mother suffered from genital tuberculosis while pregnant, the transmission of the disease under such circumstances to the developing embryo being, one would say, a very likely accident. The fact, however, that such contamination of the embryo within the tissues of the mother happens so seldom, shows with what difficulty the transference is effected. It has been asserted, but proof is certainly wanting in confirmation of the allegation, that the ovum may become infected through the spermatic fluid emanating from a person suffering from tuberculosis of the testicle or its adnexa. That the ovum at the time of impregnation may become inoculated, that in a manner it may become fertilised by the spermatozoid and inoculated with the bacillus of tubercle simultaneously, I cannot imagine to be true. After conception has occurred, and the embryo is fairly advanced in development, there is a possibility of such an occurrence, but to my mind the likelihood of its taking place even then is extremely small.

Putting, therefore, the view of tuberculosis being congenital out of court, we have to fall back upon the explanation of the undoubted proclivity which the disease has to run in certain families as dependent upon one of two factors—either direct inoculation of the individual in extra-uterine life, or the inheritance of a particular predisposition, the tubercular temperament. No doubt when a member of a household becomes tubercular, the tendency to infection of other members, through family relationships, is very great, but this cannot be held to be the only cause of the disease selecting such families, and, it may be, affecting several generations in succession. Every physician and surgeon knows that the members of such families are, in a large number of instances, notable as tubercular subjects from their conformation of body and the liability they manifest to certain diseased conditions, not necessarily tubercular, but which nevertheless are sufficiently striking to confirm the suspicion of the hereditary taint. I need not enter into details of what this habit of body is characterised by; the features I am referring to are matters of daily experience with you all. My argument is that it is this habit of body, not the disease itself, which is inherited; and if you asked me in what the particular vulnerability consists, I should reply that, most likely, it resides in the epithelial protective coverings of the body being too little resistant, too easily stimulated by external agencies, too readily penetrated by the parasite of the disease. There is good reason to believe that, instead of Man being an animal very prone to tuberculosis, he is extremely insusceptible, otherwise it is difficult to explain how tuberculosis has not utterly decimated the human race. When tuberculosis is freshly introduced among savage communities, such decimation has been known to occur. Civilised nations have probably become in a manner hardened against the ravages of the parasite. There is no reason to believe, however, that any member of the human race is immune to its influence, in the sense that certain animals are immune to parasitical diseases which are peculiar to Man, or in the sense in which an animal may be rendered immune artificially. There is good reason for affirming, on the contrary, that there are few members of the human race who could not be inoculated with the tubercle bacillus were it implanted in their tissues.

The lung is the portal at which the organism usually gains

[illegible]

[illegible] found up [illegible] tuberculosis [illegible] this tubera- [illegible] there is every [illegible] generation to genera- [illegible] be taken into account [illegible] accompany the tuber- cular habit—namely [illegible] degree of colour of the hair [illegible] air in the bushy eyebrows and long eyelashes [illegible] occurrence of a lanugo-like overgrowth in tubercular children along the spine and over the legs. To my mind these all point to an anomaly of the epithelial type which is peculiar to the tubercular habit of body.

Granted, for the sake of argument, that this habit of body, [illegible] handed on from one generation to another, is the [illegible] the hereditary disposition to tuberculosis, and that it

manifests itself in certain peculiar anomalies of the epithelia, we may ask, in reference to the subject of debate, how it is that this habit of body has arisen? Where has the inherited strain come from? What is its ancestral history? Can it be generated by vicious surroundings? I question whether it can. No doubt, once in the blood, the particular habit may be fostered by every external agent which tends to deteriorate the natural powers of resistance. But will such external agencies tend to produce a particular colour of hair, a certain narrowness of chest, tallness of stature, and other peculiarities which are distinctive of the tubercular constitution? My conviction is that they will not, and that we must go much further back in the history of the human race to get at the explanation of the matter. My own impression is that these features are the lineal descendants of a variation which took place far back in our history, that the variation has occurred irrespective of surroundings or external agencies, and that its influence has been propagated in the descendants ever since. It may be a variation which is common to many races, but one which apparently is intensely hereditary. When we see how racial peculiarities are propagated for ages, how the type of character no less than the lineaments of a race continue very much the same through all time, is it very Quixotic to suppose that a certain type of constitution has in a like manner become a race inheritance? It seems to me, on the contrary, to take a concrete example, that if the spirit of commercial enterprise exemplified in that memorable transaction between Abraham and the children of Heth, concerning the purchase of the cave of Machpelah, be still the spirit of the Jewish nation, there is no reason to believe that a variation of structure predisposing to disease may not have been handed down to us from remotest times.

The second so-called hereditary disease which I wish to bring before your notice for critical examination is gout. There is no more common belief than that this disease is engendered of high living, and that, once established, it is capable of being hereditarily transmitted. In a certain sense this notion is correct, and in a certain sense it is, to my way of thinking, entirely erroneous. There is no doubt that once the gouty tendency has been made manifest by the external agencies referred to the disease will most likely show itself in

up indissolubly with organic substrata, and as these are hereditary, it follows that intellectual aptitudes are so also. As Debierre puts it—"Man thinks and acts, not spontaneously, but according to the blood which is in his veins; that is to say, according to his heredity. He thinks, he feels, he wills much more through his ancestors than through himself." Thought is nothing more than a secretion, the result of a certain metabolism; and it stands to reason that from time to time, in the phylogenetic history of a nervous system so complex as that of Man, variations in the durability and resistance of the mechanism underlying this metabolism must have taken place, as we know to have occurred in the internal mechanism of other organs of the body. Mental derangements, we know, are among the commonest of diseases. It may be that the individual is not so far deranged as to require restraint. There are minor degrees of mental derangement which may be included under the terms wrong-headed, eccentric, fanatical, or hypochondriacal, which may manifest themselves in a line of psychopathic individuals, which are quite distinctive of the type, and which may break out at a certain period, and in a particular member of the family, in one or other of the various definite modes of what is commonly termed mental alienation. All such manifestations indicate a certain diminished resistance or power of endurance on the part of the nerve-cells of the brain; and in this relationship, and in reference to what I have indicated as my own persuasion of the essential element in the heredity of the tubercular constitution, it must be borne in mind that nerve-cells are of epidermic origin, and that the tubercular constitution is frequently associated with the psychopathic. I have attempted to trace the predisposition in tuberculosis to a want of resistance on the part of the epithelia to the encroachments of the tubercle bacillus. May not there be a certain connection between the two classes of disease in respect of their representing, in reality, the same hereditary peculiarity, the same dyscrasia, the same tendency to decay on the part of nerve-cells and epithelial-cells?

I have often thought that another factor predisposing to mental derangement is, that nerve-cells show so little capability, if any at all, of regeneration. We are born apparently with the nerve-cells which will serve us throughout life. Is it a matter of wonder that frequently these break down under unusual

strain, or what is probably much more likely, as an inherited peculiarity? The germ-track followed in the ontogeny of the nerve-cells is very short, far shorter than in the case of many other cells throughout the body, and hence a state of maturity is reached at a comparatively early period, with an inclination to premature decay.

But in this class of diseases, as in others which may be considered in the ordinary acceptation of the term to be hereditary, the great question continues to assert itself, namely, whether external agencies can bring about a state of the nervous system which is distinctly morbid, and which can be transmitted for generations in the offspring. The evidence in support of the positive view of this question which is usually quoted, is that of the production and hereditary transmission of epilepsy in guinea-pigs. Brown-Séquard's experiments appeared to show that after hemisection of the cord or division of the great sympathetic in the neck, not only did the animals become epileptic, that is to say, not only could they be readily thrown into a convulsive fit, but that this peculiarity of constitution, thus engendered, could be transmitted to their young. The operation on the sympathetic brings about a trophic lesion of the eyeball which it was asserted is also transmitted. I must confess it has always seemed to me that an element of fallacy has entered into these experiments which would require to be eradicated before we can found any conclusions upon them. Have we crucial evidence to show that a mental disease may be excited through external agencies, as, for instance, by the abuse of alcohol, in a person free from any ancestral taint, and that this disease so excited can be transmitted through several generations. My own impression is that we have not; but it would be a matter of extreme interest to me, as I daresay to other members of this Society, to hear of any confirmation of the allegation? So far as I am personally informed, I feel that, in mental derangement, and in excess of perhaps any other form of disease, we have to do with an inherited peculiarity or variation, a variation which may have occurred in a far back ancestor and lain dormant for many generations, but which inevitably manifests itself under conditions of unusual external stimulation, and which is in no respect bound up etiologically or necessitated by this stimulus. The substratum which underlies the mental peculiarity is

allied to that underlying the predisposition to tuberculosis or gout, and, probably, is referable to a fault in metabolism excited, it may be by an inherent bias towards degeneration in the nerve cells of the brain, and this is eminently hereditary.

From what I have already said it will be apparent that I do not regard syphilis as a hereditary disease. Congenital it certainly is, but I fail to note that, once acquired, it can be transmitted as a truly hereditary affection. As in the case of most contagious diseases, the contagion can pass from mother or father to child, but has an inevitable tendency to die out in the early years of life in the first descendant. This is not heredity but intra-uterine contagion.

Hæmophilia appears to be, in many instances, a truly heritable disease, and so far as our lights go in pointing its pathology, they seem to indicate that it is more a disease of the blood-vessels than one of the blood. The vessels do not seem to contract as they ought to do, and their coats are peculiarly friable. One could quite well understand this peculiarity being transmitted, just as the tendency to hæmorrhoids is said to be transmissible on account of the veins concerned having unusually thin walls. I have often thought, however, that there is something more in hæmophilia than this mere anatomical peculiarity, and that there may be a close relationship between it and menstruation. The fact that it is transmitted in the male line is significant. It has often been alleged that menstruation occurs in the male as well as the female; that although there is no actual discharge of blood, there exists the monthly constitutional disturbance of the general functions in the male as well as in the female, accompanied, it may be, by rise of temperature, a tendency to undue relaxation of the blood-vessels supplying the organs of generation, and possibly also of the hæmorrhoidal vessels. There seems to me to be a good deal of truth in this allegation. May not hæmophilia be simply a manifestation of this menstruation in the male?

Many more examples might be brought forward illustrative of the thesis I have started with in my remarks, but the above will probably suffice to elicit the opinions of others.

Turning now to another matter, I would ask the attention of the meeting to the consideration of what evidence we have

of a hereditary tendency to disease becoming eradicated by inbreeding with individuals having no such tendency. How soon can we assert that the hereditary psychopathic constitution becomes eradicated in the descendants under such circumstances? Or, does it become eradicated at all? If we accept the statement that the extrusion of the polar cells with their quota of chromosomes before fertilisation occurs represents a true reduction process, that at this time a certain part of the maternal heredity is got rid of by this means, have we any reason for believing that, in course of time, a hereditary vice of constitution may entirely disappear? And here the facts of atavism come in to our aid. Polydactylism in man is supposed to be a reversion to a heptadactylous ancestor; the striping of mules is said to point back to an early equine ancestor, and horses and asses are said to contain solitary "zebra" determinants in their germ plasm; the three-toed horse anomaly is alleged to be a reversion to the fossil hipparion of the eocene period; the lanugo of the fœtus, a reversion to a hair-clad ancestor. I am told by a breeder of black polled Angus cattle that the progeny of a perfectly black polled bull and cow is sometimes a red calf with horns; indeed, unless the pedigree of mother and sire is pure for generations back, that no reliance can be placed upon the progeny they may beget. If these statements be true, what are we to say of the transmission of variations in constitution? My firm conviction is that if a vicious line is introduced it may die out, and probably does in most cases die out by interbreeding with a series of pure stocks, but that no reliance can be placed upon its not recurring atavistically, it may be, generations afterwards.

Lastly, let me refer briefly, and it must be briefly, for I am afraid I have already exceeded the limit of time put at my disposal, to the very interesting subjects of maternal impressions and telegony in disease. The influence of the mother upon the fœtus *in utero* must be very great, perhaps far greater than is generally admitted, in so far at least as nutritional factors are concerned. The subject of maternal impressions, however, is open to many fallacies, mere coincidences being interpreted as evidence bearing upon the matter. For my own part, I have little belief in maternal impressions as a cause of malformation or other peculiarity. As an example of the sort of evidence usually brought forward in support of the theory, I

may refer to the case mentioned by Hippocrates of the white woman who bore to her white husband a black child, and who explained the occurrence by there having hung in her bedroom a picture of a black man—and so on it goes. Have we any proof of the occurrence of telegony in disease? From the evidence collected by Romanes and others, there seems to be no doubt of the mother being impressed by the male element of a first pregnancy, in such fashion, that the subsequent progeny by another male partake of the features of the father of the first born, and I daresay Professor Cossar Ewart may have something interesting to tell us on this point. But what I would endeavour to elicit from the discussion, this evening, in this relationship, is whether we have anything analogous in the transmission of hereditary diseased states of body. Are there any grounds for believing, for instance, that a father with a strong tubercular hereditary history, and bearing the lineaments of such in his person, can so affect the system of a healthy mother that the children subsequently born to the same woman by a second husband, with no such strain in his blood, and no such lineaments, have the tubercular characteristics of the first husband impressed upon them? I know of no evidence bearing on the problem, but possibly some members of the Society here this evening can throw light upon it.

In conclusion, allow me to formulate the substance of my remarks in the following synopsis:—

(1) There is no evidence proving that diseased conditions of body, excited by external agencies, using the term in its broadest sense, can be transmitted hereditarily through generations.

(2) That the various hereditary tendencies or predispositions to disease of the hereditary type have arisen as variations in the germ-plasm.

(3) That these predispositions to disease probably extend far back into the history of the human race, and break out only occasionally, in accordance with the laws of atavism.

(4) That external agencies are merely the means of bringing them to light.

(5) That there is little if any reliance to be placed in the evidence bearing upon the influence of maternal impressions.

(6) That there is no reason to believe that telegony may

not prevail in the case of hereditary predisposition to disease as it evidently does in regard to other characteristics.

Discussion

Dr Clouston, in opening the discussion, said that Prof. Hamilton had introduced a very vast and intricate subject, indeed a series of vast and interesting subjects, which to discuss thoroughly would take at least a winter session of this Society —subjects about which half a dozen treatises could be written. He was sure he expressed the minds of members of the Society that they were all deeply indebted to Prof. Hamilton for the way in which he had brought the matter forward.

With regard to the theoretical view of heredity now most generally accepted among biologists—that of Weissmann—it seemed to him that Weissmann had made three very striking admissions in regard to his general thesis that the effect of acquired characters cannot possibly be transmitted. In the first place, he admits that the germ plasm can be infected by such a disease as syphilis, so that thus the disease may pass from parent to child. His second admission is that the law of non-transmissibility of acquired characters does not apply to the very lowest organisms. The third admission is most important to medical men, who, he assumed, looked upon the matter from a practical rather than a theoretical point of view. It is that environment, especially climatic conditions, has the power of influencing the nutrition of the germ cell itself, and so altering its constitution that you may have an organism produced which is in some respects different from its parents. There was no doubt these admissions went some way to satisfy the universal experience and the universal instinct of the profession as to the heredity of disease.

In regard to the psychopathic constitution, he thought there was no doubt on the part of all who had looked into the subject, that it was well proved to be most hereditary. For two hundred years or more one of the royal families of Europe, whose history is well known, have undoubtedly exhibited this constitution in all its enormous varieties. Confining his remarks to the tendency of hereditariness in the neuroses, it always seemed to him to be an important admission on the part of Weissmann that environment and evil climatic conditions, and among these we must include evil dietetic conditions, may affect the germ plasm. Looking at heredity, particularly from a physiological and pathological point of view, what is the most important thing that good heredity can give the individual? He would say, in the first place, the power for the individual—the product of the germ cell and the sperm cell—to integrate,

to develop and maturate on normal lines, from the moment of the first contact up to the full development of the individual. Assuming that you can have a certain debasement of this power, such an alteration in the nutrition of the germ cell as to affect it unfavourably, had we not in this way a practical explanation of a vast number of the neuroses? Taking the neuroses one by one, it would be found that by far the most important arise during the period of development through some defect in the maturation of the cells and of the tissues. In the case of epilepsy there was no doubt whatever that at least three-fourths of all epilepsy occurs during the period of development, and this was also the fact with regard to St Vitus' Dance and the great crowd of other nervous diseases, irrespective of the great number of congenital defects, idiotcy, imbecility, and teratological defects generally. In regard to insanity also, there was no doubt that mental alienation occurred in its most marked and characteristic forms during the developmental period. Looking at the brain cells in the mental area, if we examined a typical cell by the three or four processes best adapted for showing its constitution, it would be seen (and he thought Professor Schäfer would bear him out in this) that there was no more integrated and complicated structure in the human body than one of these larger brain cells. The Nissl's bodies, the reticular network, the fibrillation, the peculiar structure of the nucleus and even in the nucleolus—all this made one amazed at the enormous complication of this one cell; and there are in the brain something like three or four hundred millions such. Considering the integration of such a cell from the embryo up to the age of twenty-five, and thinking first of its marvellous structure, and secondly of its function, mentalisation in its numerous forms, and the connection of mentalisation and emotion with nutrition and with all the bodily functions, we can imagine how very small a deficiency in the nutrition of the germ plasm—how very small a change may mean some arrest, some debasement, or stoppage of the developmental process in groups of these cells; and such a small change he assumed to be possible on Weissmann's admission. In this he thought we had a theoretical explanation of certain hereditary defects. They were defects in the development of the nerve centres, arrests of maturation of the cells that constitute these centres. In this way we had a basis for the heredity of a large number of the neuroses.

There was no doubt whatever of the non-resistiveness of certain tissues about which Professor Hamilton had spoken. Take for example ringworm. It occurs in the child simply because its epidermis cannot resist the spores, while that of the grown-up individual can do so. Applying the same principle,

he thought we should find that a very slight change indeed in the resistiveness of the brain cell would explain the well-known want of resistiveness to the numerous mental and bodily disturbing influences that act on the developing brain cells and cause adolescent insanity and other neuroses. Looking at the influence of the sexual and reproductive instinct, it was known that at a certain period of development there is an enormous strain on the constitution of every girl and every lad. Assuming that we had a slight alteration or even a germ infection of the germ plasm causing an arrestment of the developmental process; we had, he thought, an explanation in that way of a hereditary tendency. Certain individuals had weakness in the nutrition of the germ cell, and on account of this had not the power to develop into full maturity, this defect recurring in successive generations.

Then taking the other process—that of decay—the normal physiological process of growing old, coming to an end, and dying; we know that in certain families there are many individuals who become insane at the turn of life, as they become old, simply from an unphysiological method of nerve disintegration. From this cause there was a whole set of neuroses of what one might call the decadent period. Assuming that through defective germ plasm the brain cells are deficient in another great power of heredity, viz., that of carrying the individual physiologically through his decadence; there was here a theoretical explanation of the heredity of these diseases of the decadent period.

With Professor Hamilton, he did not believe, nor did he think anyone did, that mere personal mutilations, or the effects of surgical operations could be transmissible. Nor did he believe in Brown-Sequard's experiments, and he thought that Weissmann upsets to a large extent the conclusions that were founded on these experiments.

With regard to the alcohol habit, in which they were all profoundly interested, there was no doubt as to the actual fact, whatever might be the explanation, that in certain families there runs a tendency to the abuse of alcohol. He would so far agree with Dr Reid, writing lately in the medical journals, that no man has a born tendency to take whisky; but he thought a great many men unfortunately were born to what might be called a lack of higher inhibitory mental powers. He held that this was strictly analogous to the lack of developmental power in the man who was affected with adolescent insanity. There existed no doubt in these individuals a morbid reactiveness to the alcoholic stimulant. A young man with this highly neurotic tendency taking two glasses of whisky may go clean off his head, and he would lose mental power and strength of

mind with each repetition of the dose; and would settle down at length into a dypsomaniac of the very worst class, viz., one that begins during the period of development—a class of which he was sure all present must have had experience as a nuisance to themselves and to the world. It was not the craving for alcohol that was inherited, but a general psychopathic constitution in which the alcoholic stimulus is an undue stimulus, and the mental control deficient. He thought it must be admitted that it was essentially a hereditary weakness in the brain reactiveness to alcohol; some people being able to take a large amount with impunity, others not being able to take a drop with any impunity whatever. A neurotic heredity is thus seen to resolve itself into general morbid tendencies rather than direct proclivities to special diseases; and this seems quite compatible with the Weissmann theory of heredity.

He was struck with one of Professor Hamilton's observations —that with reference to the Jews. Now in Jerusalem he once counted four distinct types of Jews—all quite different—so that he thought the Jewish element of permanence was really not so great as has been supposed. He saw the common type of dark complexioned, hook-nosed Jew; the red-haired, turn-up nosed Jew; the Georgian, tall, proud-walking Jew, fully armed with pistols and poniard; and the Indian Jew of the Hindoo type.

DR JOSEPH BELL was not prepared to take part in the discussion, but thought that in the course of a few minutes he might bring out that there was some ground for the belief in heredity from the surgical as well as from a public point of view.

A Board of Insurance would not pass a man who had three aunts and two uncles dead of tuberculosis without a certain amount of loading. If his ancestors have had cancer, they will take a similarly serious view of the case. He thought, however, that tuberculosis, syphilis, and cancer might be put aside as not being hereditarily transmitted diseases, but which might be transmitted in the germ cell to the germ cell as has been mentioned.

There was one little point in surgery which always struck him as interesting, and about which he had no doubt; that was the hereditary tendency in certain families to the formation of those small cystic or encysted tumours called wens on the head and upper part of the body. On every opportunity he had inquired of the possessor of one of these wens as to their occurrence in his ancestors, and had nearly always found this to be the case. Now the child was not born with one of these tumours, but what was transmitted was the hereditary tendency,

some defect in the epithelium or in the sebaceous follicles, which at the age of thirty-five, forty, or forty-five, began to show itself in a large number of members of certain families. In connection with these matters of heredity there were, however, certain fallacies. If a woman thinks she is going to have cancer, she will be pulling about her breasts and brooding over the matter, and will be much more likely to get the disease than if she did not think of the matter at all. He had seen many cases in which a perfectly healthy woman had worried herself into the belief that when she came to the age of forty-five or fifty she would have cancer, because some aunt or other was afflicted at that age—at anyrate, something was produced which had to be removed, and which recurred and gave a lot of trouble.

There was a great tendency towards the inheritance of certain abnormalities of development, especially to hare-lip and cleft palate. But even here there might be a fallacy, as was instanced by the case mentioned by Professor Haughton of the young cubs which all died of hare-lip, but which was at length explained by the fact of the keeper having fed the lioness while in the family way on tit-bits, but without bones; and when the bones were subsequently supplied, the *hereditary* tendency to hare-lip entirely disappeared.

The whole subject was full of fallacies; he had, however, never heard a more interesting paper on the subject than that of Professor Hamilton, nor one which had introduced such a vast amount of discussible matter.

Professor COSSAR EWART would first congratulate Professor Hamilton on having arrived at a very orthodox view of the subject. He thought that on nearly every point Professor Hamilton's opinion was, from a biologist's point of view, as nearly as possible orthodox. On the other hand, he should say that Dr Clouston represented the opinion still held by the vast majority of medical men—a belief in the transmission of acquired characters. In Dr Clouston's remarks, one felt that all through he argued in favour of such transmission. There was, however, no single point that he could take hold of to controvert; he would therefore leave him to be dealt with by other speakers.

With regard to Professor Hamilton's first point—that in the case of gout, tubercle, and other diseases, they referred to a long time ago variation, which led to the appearance of gout in one family and tubercle in another; and that all the tubercle in the world, or most of it, has been inherited from previous variations. I don't think it is necessary to go so far back for either gout or tubercle. The variation that occurred a million years ago or a thousand years ago might also occur to-day. There was nothing

that struck him more than that variation was enormously common. It was the easiest matter to produce a perfect epidemic of variation. Domestic animals reproduce themselves with great uniformity if kept apart; but the moment one mixed up two different races, strains, or breeds, one did something that was difficult to put in words, but the result was what had been best described as an "epidemic" of variations. In the human family there was a constant mixing up in all parts of the world, and the result was always a certain amount of variation. Hence he thought that the particular condition of things which predisposes or is favourable to the growth and development of the germ of tubercle might appear in any country, and might go on reappearing for ever.

Then coming to the question which ran through all this discussion—the influence of the surroundings or environment on the germ-cells, he understood that Dr Clouston contended that Weissmann admitted that the germ-cells might be altered by environment. He was quite sure of this, that Weissmann holds to the continuity of the germ-plasm; that the power to vary we find in the higher animals was acquired originally by their remote ancestors; and that the variation required to produce new types is obtained by what he calls a "reduction" of the germ-plasm, *i.e.* by a part of the nucleus of one germ-cell (the ovum) being got rid of to make room for another germ-cell (the sperm) having different potentialities. He (the speaker) thought Weissmann would not admit that the germ-cell is directly influenced by environment—that the environment is the actual cause of variation.

All the work which the speaker had been doing lately pointed to the fact that the germ-cells are excessively sensitive, but not to the fact of there being any transmission of acquired characters. If, for example, he mated a very young pigeon with an old pigeon, taking those breeds that have peculiar characters such as frills, hoods, etc., the first produce of the practically immature pigeon are all perfectly smooth-headed and smooth-breasted. But the next set of young, when the bird is in good form and feather—has fully reached maturity—may have all the characteristics of the parent. That meant that the first germ-cells are in an immature condition; so feeble or impotent that they are incapable of handing on the highly specialised characteristics of the parent. Give the germ-cells time and they will become so prepotent that they may hand on all the peculiarities even when the pigeon is crossed with another breed. In the same way if a rabbit that is still immature is put to an old buck—the buck may serve her, but the germ-cells being immature, there is for a time no ovulation. The sperm-cells lie waiting, sometimes for over a week, and the moment

ovulation takes place the eggs are fertilised; and in this case the young all take after the male. If on the other hand a doe is served, not at the right time, but a week or ten days after, when the next young come they are all exactly like herself. In the first place the eggs are hardly ripe, in the other they are over-ripe. He might give many instances to show how much the maturity and ripeness of the eggs determines the condition of the adults developed from the eggs. This was of course altogether different from Weissmann, and he thought it would, when thoroughly understood, enable those who cannot live without the transmission of acquired characters to feel happier.

Professor Hamilton had, all through his paper, escaped the fallacies which had so long prevailed; but until others had reached the same stage there was not much prospect of progress.

He might, perhaps, say a few words on another subject, viz., reversion. It had been recently alleged that there was no such thing as reversion. The subject was originally discussed in a scientific way by Darwin, who thought that reversion had resulted from a sort of antagonism. That reversions occurred the speaker had no doubt. By crossing a pure white fantail having thirty feathers on its tail with a cross-bred pigeon, he had obtained a slaty-blue bird with only twelve feathers in its tail, which closely resembled the wild blue rock pigeon.

In the same way he had evidence of reversion in the Equidæ. In his zebra hybrids he expected to get something between horses and zebras, but had got something in its colouration very unlike either a horse or a zebra. The question arose—was it a new creature or an old creature? By making careful investigation as to stripes, etc., he came to the conclusion that his hybrids in their markings were a restoration of an extremely old type of horses. This had been verified to a certain extent by the discovery in Tibet recently of ponies almost as striped as some of the zebras. The Himalayan ponies and certain ponies found in Norway had characters which agreed in a general way with the hybrids—so that in these hybrids he believed we had again a very marked instance of reversion.

Dr J. W. BALLANTYNE said that he might best begin his remarks by referring to one point in Professor Hamilton's communication with which he could fully agree, namely, his disbelief in the potency of maternal impressions to cause conditions in the fœtus resembling the impression. At the same time he thought it might be said that to whatever extent we believed the mind capable of influencing the state of

a part of the body, to that same extent, or to a degree rather less, the mother's mind might influence her parasitic growth (*i.e.* the fœtus *in utero*). But this amount of belief would of course vary very much in accordance with the elasticity of our belief regarding the influence of the mind over the body. Personally he lamented very much the intrusion of the subject of maternal impressions into biological problems, and more especially into antenatal pathology; it had kept that subject back, and had spoiled many case records which would otherwise have been most valuable.

Having agreed with Professor Hamilton on this point he was now forced to differ from him on two others. He did not think that we could separate off clearly the diseases of the fœtus due to intra-uterine infection from the hereditary diseases. Take first the case of small-pox, about which there could be no difference of opinion. When a pregnant woman had small-pox her fœtus *in utero* might take it. Manifestly the fœtal variola was not passed to the fœtus by heredity. Take another condition which could not be so easily divided off—fœtal tuberculosis. He thought there was no doubt that fœtal tuberculosis truly occurred, but very rarely; it was not to be expected that it could occur often, for the fœtus cannot be infected through the air (*i.e.* by the lungs) but only through the umbilicus; and as it was seldom that the tubercle bacilli were in the mother's blood, it was seldom that they reached the fœtus. Cases of fœtal tuberculosis had been reported, and he thought that (*e.g.*) the one described by Auché and Chambrelent formed an absolutely complete proof. In that case the mother had generalised tuberculosis, and tubercle was found in large amount in the fœtus in its liver and spleen (just where it was to be expected, considering that it entered by the umbilical vein), and to a small amount in the lungs. It was found also in the heart, in the form of tubercular endocarditis. Now, here was one of the points which he wished to emphasise: heart disease was one of the conditions admitted to be sometimes transmitted hereditarily. A mother with tuberculosis might give birth to a fœtus not with tuberculosis but with a cardiac morbid condition. He was quoting Hanot when he said that it had been advanced that the offspring of tubercular parents might escape tubercle by having a cardiac malformation or a congenital condition other than tubercle; it has been said that in this way the child might get rid of the heredity of tubercle. Was it not possible, however, that this was a case in which the disease was not transmitted from mother to fœtus in the same form, for it did not follow that a mother must transmit the disease to her fœtus in the same quality as she had it herself? She can and does transmit something to the fœtus which is not exactly the

same as she has herself. For instance, there was Bidone's case of erysipelas in the last days of pregnancy: the mother had it showing itself in the usual way in inflammation of the skin, but the fœtus was born without any signs of erysipelas in its skin, but it had endocarditis and on the valves of the heart streptococci were found, and streptococci were also discovered in the placenta and in the mother's skin. Could it be doubted that the erysipelas in the fœtus was due to the streptococci, and took the form of endocarditis instead of dermatitis? To carry his argument a little further, it might be maintained that all the things he had named were not *hereditarily* transmitted but by infection; but could any line be drawn between things so transmitted (taking into account the peculiarities of the fœtus and the change in the nature of the handed-on morbid process caused thereby) and the transmission of toxines which did not produce any special disease but simply made the fœtus less well nourished and more likely to take infections?

To go a step further, the embryo might be cited. The modifying effect of environment on the development of the embryo was one of the most clearly established of the phenomena of teratology; malformations could be thus experimentally produced (those at any rate characterised by arrest of development). Toxines of various kinds, hydrocyanic acid very powerfully, nicotine, the alcohols in the degree of their toxicity, these when injected into the hen's egg caused teratogenic effects. In the human subject there was evidence of the clinical kind to support this, for malformations were found more commonly in the descendants of individuals who had saturated themselves with alcohol or had suffered from lead poisoning or other toxic condition or from the infections. Now many of these things were transmitted hereditarily. So he had passed in his argument from small-pox, which no one regarded as transmitted by heredity, through a series of connecting links to conditions such as malformations which were in many cases markedly hereditary in their transmission. Further, the malformations agreed with the neuroses and with gout, rheumatism, and other things, in obeying the same laws in heredity, such as family prevalence, dissimilarity, etc. These laws applied to the various morbid phenomena of antenatal pathology, viz., malformations, monstrosities, prematurity, dead-birth, congenital debility, twins, etc. So the first point in which he differed from Professor Hamilton was that he thought it not possible to draw a sharp line between what everyone admitted to be a fœtal disease (an intra-uterine infection) and a hereditarily transmitted condition.

In the second place he differed with respect to the value of the experiments disproving the transmission of acquired char-

acters. He thought that so far they had not been tackling this subject in the scientific sense, for they had apparently always begun with the idea that the character to be acquired must be acquired after the birth of the individual. That did not seem to him to be the proper scientific way of looking at the subject. The individual lives before he is born, and it may be that experiments failed to give positive results because they (*e.g.* mutilations, etc.) were begun after birth. To test the matter fairly, ought not the processes to be commenced while the embryo or fœtus was still in the uterus of the mother-animal?[1] If this were done, positive results might be looked for. But apart from this there were Kohlwey's experiments on pigeons: he cut off the posterior digit of the foot, and the mutilated bird got in the habit of turning the fourth digit backwards and using it in perching; he got no descendant of these mutilated birds without a posterior digit, but he got a descendant of one of the pairs with its fourth digit turned backwards like the first. The mutilation was not transmitted, but the physiological adaptation to meet it was.

How were we then to get at this problem of heredity, for there was no possibility of avoiding it? Attempts had been made, so to speak, to outflank its position, and the solution of heredity had been sought among the unicellular animals. It had been attempted to circumscribe the field of inquiry to characters transmitted by sexual generation, and the problem had become only more complex. He thought we must just keep on driving straight ahead, passing towards heredity through the series of antenatal morbid phenomena which lead up to it. In that way one would get back to the germ, and by considering in turn the various links he thought that at length a solution might be found. In conclusion he might state that to his mind two of the special problems of heredity were most inscrutable: one of these was telegony; the other was expressed by Montaigne in the sixteenth century, when he puzzled over the riddle of why he should develop a stone in the bladder at the age of forty-five as a legacy from his father who, however, only developed the same thing when sixty-seven years old, or twenty-five years after his son had been born.

Professor STEWART STOCKMAN said he would only take up a few minutes. In the first place, with regard to tuberculosis. Not so long ago, when he was a student, he was taught to go into a byre, look at a cow, and say whether she was likely to become tuberculous. Since the introduction of

[1] As Charrin and Gley have done with blue pus and rabbits.

tuberculin it was now easy to diagnose what are called occult lesions. With the help of tuberculin it had been found that really these cows were not predisposed to tubercle—they had already got it: He had often wondered what the effect would be of submitting the human race to the tuberculin test. He thought a great many of those individuals who were looked upon as predisposed would be found to be really tuberculous.

As regards congenital tuberculosis, his opinion corroborated that of many others. Nearly every week for the last eight years he had gone with his class to the slaughter-house to examine diseased organs and carcasses; and he had been on the look-out, and the inspectors had also been on the look-out, for cases of congenital tuberculosis.

They in Edinburgh slaughter something like six thousand calves a year, and he had found only one case of tuberculosis among them that was undeniably congenital. Of the cows slaughtered, at least twelve per cent. were tuberculous. This was a low percentage, because they were so strict in Edinburgh that most of the doubtful cases were sent to be slaughtered elsewhere. The general average would be about twenty-five per cent., but in some herds it mounts to eighty per cent. of the effective.

Now, if you take the bullocks and heifers, the tuberculous animals do not amount to one per cent. Surely, if even a predisposition were transmitted, it ought to be to the bullocks as well as to the cows. The question was really one of external predisposing causes.

With regard to the so-called predisposition to tubercle, there was one point to which he thought too little attention had been paid—the influence of heredity, not upon the patient, but on the tubercle bacillus itself. The bacillus taken from one species often fails to affect another. For example, they all knew that it was very difficult to give human tuberculosis to fowls, but occasionally a fowl may be inoculated successfully. It has been shown lately that if the tubercle bacillus from a human being be grown in glycerine broth, put in a collodion capsule, and placed in the abdominal cavity of a fowl (the collodion prevents phagocytes acting on the bacillus), it after a time acquires the characters of the avian tubercle bacillus, and in the course of some months you obtain a variety which is deadly enough to the fowl.

He had inoculated horses and donkeys with different kinds of tuberculosis, and others had done the same. The first impression was that the horse was very insusceptible, and the donkey absolutely immune. The horse is now known to be susceptible, though no one had ever pointed out a horse that was likely to become tuberculous; the study of equine tuberculosis dates from the discovery of the tubercle bacillus. If one

take the bacillus from the cow and inject it into the donkey, lesions are produced, but these are cured in about a month; and in his first experiments he thought the view was quite correct that the donkey was not susceptible. It happened, however, that one day the tuberculous spleen of a horse was sent him for examination. With material taken from this he inoculated a donkey, and the donkey died of tuberculosis in the ordinary way. He thought that when talking about one species being predisposed and another not, we should remember that if the bacillus does not act on this or that species, it may not be due so much to the species as to something in connection with the bacillus.

As regards cancer, he had never heard it seriously said among veterinarians that it was hereditary. Yet they saw a good deal of cancer. No one, however, had observed a series of cases in respect to its being hereditary in animals. Glanders also was a disease which affected horses. In its course it was rather like tuberculosis. It was now known that it often ran a very chronic course, and that the lesions might remain for a long time occult; but it had never been seriously said that it was hereditary. When he heard Professor Hamilton express the view that there was no hereditary tendency in the case of syphilis, he thought he would like to have it explained why this should be so in regard to syphilis and not with regard to tuberculosis?

Lastly, there was one nervous disorder in horses, termed "roaring," *i.e.* paralysis of the left recurrent laryngeal nerve. It was a rank heresy among the majority of horsey men to say that roaring was not hereditary, and yet there were far more tangible explanations of this affection. The famous horse "Ormond" was a roarer, and his sire was a roarer. He was sold for twelve thousand pounds because he was of a roarer strain. He has been sire to a number of colts in this country. Some have become roarers, others have not. It has been said by veterinarians that in such diseases as strangles, influenza, and suppurative diseases of the throat, the bacterial toxins may produce a condition of the nerves which results in roaring. There are also other good explanations of the condition. In the Argentines, where Ormond first went, it is said that none of his progeny have become roarers. Evidently the hereditary influence plays only a feeble part, if it play any at all.

Dr JAMES observed that it had been stated by a great man many years ago that while philosophers and statesmen were cogitating as to how the world should be governed, hunger and

love were performing the task. He thought it very fortunate indeed that humanity out of instinct and common sense should know how to conduct a good life without getting any assistance from scientists in the way of physiology. It was also a good thing for humanity that it could go on reproducing itself to good purpose without scientific aid as regards heredity. He said this because within the last few years he had heard a great deal coming from scientists which was against instinct and common sense, and it would be very bad for humanity were it to adopt these dicta practically. He referred in this connection especially to the statement that acquired variations cannot be transmitted, and to the statements that we have heard lately that much less importance should be placed upon heredity than common-sense people are inclined to allow.

Of course they knew that according to the Weissmann theory acquired variations could not be transmitted. But although Weissmann had begun by saying this, when they looked into all that had been written by him subsequently, they could not but conclude that what he had said after all was nothing very different from what had been said before him. Weissmann seemed to regard a variation rather as a development of something that was there before, than as the occurrence of something new. But is it not the case that evolution means simply the better adaptation of the organism to surrounding conditions? Hence in every generation a new step in progress is, as it were, made. Dr James remarked next that he was not going to follow Weissmann in his metaphysical processes, but he would just quote a sentence which he remembered to have read in one of Herbert Spencer's books. At least he thought he had seen it there. This was that, amongst living beings, the conditions which favoured growth and development were precisely the conditions which, given time enough, would bring into existence.

As regards examples of the non-transmission of acquired variations which had been brought forward, he could not help saying that to imagine they proved anything was simply childish. For example, as regards mutilations.

Certain tribes of Indians had for years, by pressure in early life, flattened their heads. It was said, "Why was not this perpetuated?" The Chinese women had for years distorted their feet. Why was not this perpetuated? Well, in the first place, the years during which those mutilations had been going on were as nothing compared to the duration of life of human beings on the surface of the globe. But further, and he thought that this was the better answer, Nature was not a fool, and Nature, in order to enable human beings to rise in evolution, brought about the elimination of the unfit as well as favoured the survival of the fittest. Nature was not such a fool as to perpetuate

flattened heads and distorted feet, but had eliminated or would eliminate the people who practised those mutilations.

Brown-Sequard's experiments on the epileptic guinea-pigs, and the observations on pigeons which Dr Ballantyne had quoted, were, if corroborated, of value as against the Weissmann theory. But Dr James contended that laboratory and experimental work were not likely to give valuable data in connection with the transmission of acquired variations. At any rate, as yet he held that they had not afforded much information. On the other hand; Dr James held most emphatically that doctors in practice had much better opportunities of forming an opinion on this point. Doctors had opportunities of examining for themselves, and of tracing variations in a way that the laboratory scientist never had. He remembered hearing it said by one of his personal friends that, at a recent meeting of biologists, all had declared in favour of Weissmann's theory, except a few doctors. His contention was that the doctors were probably the individuals best capable of judging.

He wished now to explain what he meant.

In medicine it is an old idea—and he thought there was a great deal of truth in it—that the immunity which an individual acquires as the result of having a disease such as typhus, scarlet fever or small-pox, may be transmitted. Personally, he could say that the most malignant examples of small-pox and scarlet fever which he had seen were in the Edinburgh Infirmary, in people who had come in from the out-lying districts of Scotland, where these diseases had not been seen for generations.

As regards syphilis it had often struck him that some of their surgical friends have opportunities which should not be neglected. A man has had syphilis and has been carefully treated. His offspring do not get syphilis—but are those offspring less easily acted on by the syphilitic virus than ordinary individuals, or are they not? We know if a man has acquired syphilis—though the wife may not get the disease—his child may be born syphilitic. The mother in this case is immune (Colles' law), and may not such immunity be transmitted?

Again, do those individuals whom we see presenting the marks of congenital or hereditary syphilis take the disease if exposed to infection in as virulent a form as those who have not had it in this way?

Next, as regards alcoholism. Dr James was strongly of opinion that alcoholism in a parent produced diseases of a good many kinds in the children in this country. Of course it might be said that the alcohol which the parent had taken had simply brought into prominence what was there before. He was not going to enter into this precisely, because he believed it could do

no good, but he held that whether the predisposition to alcoholism was in the parent or not, the practical indulgence in it was very injurious to the offspring. For example, he had, in looking up some of his case books, gathered together some statistics in connection with two diseases, epilepsy and phthisis. In order to limit himself to heredity, he had taken only the epileptic cases under twenty years, and the phthisis under thirty. He found, curiously, that a phthisical family history prevailed in the same proportion in the epileptic as in the phthisical cases. But he had no hesitation in saying that in a large number of the epilepsy cases in children in which it was stated that the parents were quite healthy, one parent, usually, of course, the father, was often found on inquiry to have been alcoholic. Again and again he had found that an alcoholic parentage with perhaps a fall or an injury to the head in childhood, had produced epilepsy, and he looked upon the epilepsy in such cases as an evidence of the transmission of an acquired variation.

Another disease which he had looked into was gout. An insurance friend of his had once informed him that the heredity of gout had been much over-rated, because it had been found that individuals who had been rated up for a family history of gout, had proved to be specially good lives. Now, at first sight this might seem to detract from the importance of heredity, but when we look at it properly, it proves rather how prone we are to draw false conclusions. That individuals with a family history of gout should often prove to be the best lives we can quite easily explain. A gouty heredity means specially good digestive and assimilative powers; and specially good digestive and assimilative powers very often means that organic sensation of well-being which is apt to lead to excess. As Sydenham said long ago, "More wise men than fools have suffered from gout." Now, the men who insure their lives are usually men who have, and can exercise self-control. Hence a gouty heredity in connection with insurance means specially good digestive and assimilative powers, plus the self-control that prevents excess. The statement, then, that heredity is of no importance, because lives rated up for gout often prove the best lives, is a false one. What we should say is that heredity is important, but that we must make certain that we are interpreting its data correctly.

Professor SCHÄFER said he would content himself with saying a word or two on certain special points on which he had been appealed to. With reference to the complexity of the nerve-cell of course he agreed entirely with Dr Clouston as to its complex structure, but that fact he thought

rather accentuated Professor Hamilton's position. No doubt with the early maturity of the nerve-cell it was hardly conceivable that it would transmit any acquired character. He was quite sure that Professor Hamilton would admit that the discussion was to a certain extent a groping in the dark, because we were completely ignorant as to the causation of disease except such diseases as were due to parasites. He thought he was right in saying that we knew nothing as to the actual causes of other diseases. It was, therefore, very difficult to say what they were dealing with when they talked of heredity in disease.

One word with regard to Dr Ballantyne's proposal to make experiments on the fœtus *in utero*. Of course he must be aware that such experiments have been and are being made at the earliest possible stages of development. With frog embryos, for instance, many experiments have been made on the first cells produced from the egg. (Dr Ballantyne agreed that this was being done: he understood with positive results as to the transmission of acquired characters.) The speaker only wished to point out that there was no difficulty in experimenting upon the earliest stages. As to the guinea-pig experiments of Brown-Sequard, he might say that, at the suggestion of the late Mr Romanes, Dr Leonard Hill made a certain number of similar experiments in his (the speaker's) laboratory in University College, but so far as they went it was not found that the guinea-pigs in which this neurosis had been artificially produced transmitted it to their offspring.

Professor HAMILTON, in reply, said he was sure that Dr Clouston must have had a large experience of cases bearing either for or against the question of the acquisition and transmissibility of mental diseases, and from the tenor of his remarks he was led to believe that he favoured the view that mental disease, in the ordinary sense of the term, may arise from external agencies and be transmitted. With regard to the Jewish types, he was quite right—anywhere on the Continent you might see different types of Jews—but anyone who had had sufficient experience could pick out these to be Jews. One inherited Jewish characteristic is the mental proclivity to commerce which has continued for all time. There are also bodily characteristics in the way of the thick under-lip, and a peculiar conformation of the legs which mark them off.

With regard to Professor Cossar Ewart's remarks, he was pleased to find that, looked at from a biological standpoint, what he had said had been so orthodox; he had been prepared to hear his ideas characterised as extremely heterodox.

Referring to Dr Ballantyne's remarks about congenital tuberculosis, there might be an element of truth in what he said. It was a well-known fact, which he had verified all his pathological life, that a person suffering primarily from valvular disease of the heart seldom, if ever, died from pulmonary tuberculosis. What the explanation was he did not know.

There was certainly a difficulty in separating the congenital from the hereditary, but he thought we might say that a condition is hereditary when we find it pass through generation after generation; not merely through one generation, or even a couple, but where the tendency is inherent, like the colour of the hair, or shape of the nose, though perhaps skipping a generation now and again. Of course, the infection of a child with syphilis while in the uterus is not heredity, and he thought that perhaps Dr Ballantyne might have misunderstood what he had said regarding this.

Touching upon the early experiments *in utero* advocated by Dr Ballantyne, Professor Schäfer had in a manner anticipated what he had to say in reply. The beautiful experiments on merogony carried on by Roux and Delage upon the ova of frogs and echinoderms might be mentioned in this connection. These experiments went to prove that the ovum might go on developing into a perfect larva, even though a number of the early blastomeres had been removed; it was only occasionally that any deformity was met with. Even this radical method of applying external agency had comparatively little influence on normal development.

Professor Stockman had contrasted the difference in frequency of occurrence of tuberculosis in cows and bullocks. He would point out that cows were under different conditions from bullocks, which, it might be, predisposed the former to the infection. The cow was kept in a warm, close, stuffy atmosphere, while the bullock might be, for a considerable time, in the open; and at the same time, the cow, through excessive discharge of milk, gives off from its body a large amount of nourishment likely to impoverish its system.

On the alcohol question in relation to insanity, there were no doubt a number of fallacies. We might group with the alcoholic habit the tendency to criminality and to insanity, and regard them as manifestations of the same dyscrasia. He thought the tendency to alcohol might be looked upon as an effect rather than a cause of insanity.

Note.—The foregoing reports were corrected in MS. by the various speakers.

UNDELIVERED SPEECHES

1. By Dr WILSON, Mavisbank.—For the sake of brevity one's statements must appear dogmatic; but our special effort ought to be to keep an open mind on this question. Most of our difficulties are of our own creating, because we have not taken care to translate biological propositions in terms of physiology and of pathology.

The idea of continuity of germ-plasm is not strange and it is not relevant. All living substances are as "continuous" as the germ-plasm until they cease to be; that is, they come in direct line from ancestral substance. The essential substance of germ-cells, however, is continuous forwards as well as backwards and (on paper) is *ad infinitum*. Weismann contends that somato-plasm, the body substance, is not so, that it does not "continue" into our offspring. That may or may not be true —a question which we may waive for the present. This perpetual idioplasm idea is beside the point. The relevant idea propounded by Weismann is, that the germ-plasm which organisms convey in their germ-cells is physiologically secluded; that it is essentially immune to its environment. The germ-plasm, we are taught, undergoes no essential metabolism until the time of maturation. That *may* be so. The characteristic energy of germ-plasm *may* be quite latent for a time. It *may* be that, all that germ-plasm requires for its preservation is moist warmth, just as wheat is latent for months in dry warmth. And it *may* be that, every little while, germ-plasm maturates automatically and becomes manifest in the kinetic energy of the spermatozoa and of ova. All these propositions are still open to grave question. But, at all events, this notion of physiological isolation of the germ-stuff, and of a periodic metabolism in it of an automatic kind, is quite apart from the idea of "continuity."

Weismann admits, and everyone must, that disease has some effect on germ-plasm. The katabolism of somatic death stops the germ-stuff conveyed in our bodies. And death is a relative term. Short of complete somatic death, various degrees of change occur which affect the germ-plasm—degrees of hyperpyrexia, for example, and degrees of toxicity of lymph. But we must state the question at issue more exactly. Physicians have been at fault in not discerning variations in disease. For example, when a man with tabes begets a child with epilepsy, the physician is too ready to speak of the hereditary factor in the child's case. Many similar instances will suggest themselves—cases, not of heredity, but of variation in disease. This idea is important. Diseased organisms are apt to

breed disease, but not always, though sometimes, their own disease.

We must relinquish the idea of a hard and fast line between "acquired" and "idiopathic" diseases. All diseases are to some extent "acquired"—occasioned as reactions to environment. In some cases the agencies within the organism are of more obvious importance than in others. Let us take a case in which both factors are important—a case of tuberculosis. To obviate the fallacy of maternal infection, suppose the disease to be of the avine variety, and that the yolk is not infected. Assume that the last egg of a fowl dying from tuberculosis is fertile. Weismann would admit—everyone would—that the chick is likely not to be full-grown and robust. It will fail of "nutrition," of a full capacity for regeneration, and of normal resistiveness to environment (terms which require fuller consideration). It would appear then that this chick has an idiopathic susceptibility to all and sundry, or at least to several, diseases. But it is mere slackness to call that heredity in disease. It is equally apt to be variation; the chick turning out to be epileptic, or deformed, or liable to cholera. That is all that Weismann contends for. The disease has not bred itself. And when we come to consider human diseases, and have regard to all the possibilities of infection, we must admit that there is no proof that "acquired" diseases ever become "idiopathic." Yet, with others, I venture to believe that the last word has not, by a long way, been said upon this subject. Idiopathic diseases are, for the most part, due to premature or focal arrests of development, or excesses of development or of growth, or senility or involution in the tissues. And we may with reason hold that it is *not proven* whether, for example, an arrest of cerebral development by alcoholism does or does not induce a similar arrest (among others) in the offspring. The idioplasm of the germ-cell nucleus, as Weismann conceives it, is a molecular substance. The exigencies of debate make it necessary for biologists to whittle away the conception, and it is a fair retort to say that they are hiding a theoretical unit. Weismann would himself admit that the germ-plasm and its determinants are mystical. All delimitations of tissue, including the conception of the cell as unit, are arbitrary. There is no permanent truth, but merely convenience, in the conception of a unit and its environments. The whole organism and all nature are but parts and the relations of parts. If we push the biologist to extremes, he must eventually define the germ-plasm as that substance (in or near the germ-cell) which is independent of its surroundings. That is simply to conceive a substance as capable of what the argument requires. But physiologists will be slow—and philosophers will refuse—to

entertain the conception of a living substance which has no relations.

The invaluable contribution of Weismann to the practice, as well as to the theory, of medicine still remains—that we must look to the environment, and to the reactions, of the idioplasm to explain disease and to prevent it. That should be part of a general conception. Taking for the moment, any one organism as unit, its environment is undergoing selection, and is evolving, much more obviously and rapidly than its idioplasm. It is in the selection and in the evolution of environment that the future of medicine lies.

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2. By Dr W. LESLIE MACKENZIE. — Professor Hamilton's paper appeared to me an admirable application of the doctrine of natural selection to the problem of inherited disease. On one point—telegony—his propositions outran the evidence; for Professor Cossar Ewart has shown, in the Penicuik experiments, that the supposed proof of telegony does not stand analysis, and experiment has not yet furnished a single confirmation. The discussion, it seems to me, somewhat lost sight of the precise issue so clearly raised by Professor Hamilton, namely,—Is a disease that has been acquired by the individual capable of being transmitted as such to his offspring? Toxic diseases, which are capable of infecting the germ-plasm directly, are, of course, irrelevant to the question of inheritance. Why is there any difficulty in answering the question? Mainly because the two—or more—sides to the dispute did not make explicit their fundamental assumptions. Both sides, I think, admitted the abysmal difference between a somatic-cell, which is capable only of producing its like, and a germ-cell, which contains in itself the elements of the whole body. No one seriously disputed the propositions that the material of the germ-cell, more properly the germ-plasm, is continuous directly from person to person, and that the non-germ-plasm is not thus continuous. Further, it was assumed that the germ-plasm is the bearer of all that the child inherits from the parent. Once more, it was assumed that all the characters existing in the germ-plasm that the parent sprang from may be transmitted to the offspring; that some of the characters may lie latent; but that, on account of the continuity of the germ-plasm, these may be handed on to a third and a fourth, and an nth. generation. Then all appeared to assume that if the germ-plasm varied in any way, natural selection will at once operate to develop and preserve useful variations. One assumption, however, did not seem to be constantly present to the minds of the disputants, namely, that there is a fundamental distinction between a

variation (however produced) in an individual's germ-plasm, and a modification (produced by use, or environment, or disease) in the body (that is, the non-germinal-plasm) of the same individual. The variation in the germ-plasm will result in the production of a new character in a new individual springing from that germ-plasm. But a modification due to use, or environment, that is, a character acquired by the individual, will not be transmitted to his offspring unless it first produces a definite variation in the germ-plasm. Darwin, assuming as a fact that characters acquired by an individual were transmitted, invented pangenesis to show how the transmission was possible. But Galton found reason to believe that pangenesis, that is, concentration of representative gemmules in the germ-plasm, was mythical, and also found reason to doubt whether acquired characters were inherited to any but an extremely small extent. Weismann drove the analysis further, and not only showed that many acquired characters supposed to be inherited were not inherited, but that the thorough-going application of natural selection made the hypothesis quite unnecessary. Many difficulties exist on both sides; but in the problem of hereditary disease, it is, I submit, illegitimate to assume that the inheritance of acquired characters has been proved. On the contrary, it remains entirely unproven. So far, experiment has failed to make it even predominantly probable. By "acquired" is to be understood "acquired by the parent during his own life-history." From conversation, I gathered that some understood "acquired" to mean acquired by the germ-cell. The germ-cell, or germ-plasm, is an organism within the body-organism. Like cell organisms, it depends on its environment (the body) for nurture; but nurture affects function and growth, not structure.

Consequently, it is a very "cheap" and, only in appearance, a very simple assumption to say that any structural modification in the germ-cell's environment (the body) will create in the germ-cell such an alteration that when the germ-cell grows to an adult, that adult will have the same structural modification as the parent. But to judge by the instances produced at the discussion, the inheritance of modifications acquired by the parent is a superfluous assumption. In speaking of Weismann's "admissions," Dr Clouston left on one the impression that he considered the admissions somewhat inconsistent with the fundamental assumption of the continuity of the germ-plasm from generation to generation. But when Weismann admits the "transmission of disease," he means such diseases as may infect the germ-plasm; but, as Prof. Hamilton showed, the direct infection of the germ-plasm by a poison circulating in the body is quite irrelevant to the question of inheritance. Weismann would not, I take it, admit that (say) cardiac hypertrophy due to over-work

would produce congenital cardiac hypertrophy (or even a tendency to it) in the child. Of course, the constitution that made the father liable to hypertrophy would also make the child liable, but this is inheritance of a constitutional (non-acquired) character—a thing no one disputes. Then "the laws of transmission do not affect the lowest forms" is said to be another admission. I assume the general accuracy of the statement. The explanation is simple: in the unicellular organisms there is no division into germ-plasm and body-plasm. There is, consequently, no heredity in the sense applicable to higher organisms. In the lowest forms the whole body of the parent divides into two daughters. But, in the higher, only a minute fraction of the organism is set apart for reproduction.

Again, a third admission is that "the environment, as climate, has the power to alter germ-cells." Prof. Cossar Ewart corrected this statement, which certainly does not completely represent Weismann's doctrine. The word "alter" is ambiguous; it may mean alter functionally, or structurally. The germ-cell is, of course, affected by its own environment, as I have said above; but Dr Clouston seemed to indicate his belief that climate might produce in the germ-plasm directly elements that did not before exist in it, so giving rise to new variations in the germ-plasm. This proposition needs a vast amount of proving. I have not found Weismann maintaining it, but he admits that, in certain butterflies, climate may stimulate the growth of particular elements of the germ-plasm—a very different thing. None the less, Dr Clouston is, I think, right in concluding that Weismann's hypothesis includes everything essential to the explanation of hereditary insanity. I felt, however, that in speaking of the "royal family insane for three hundred years," Dr Clouston seemed to suggest the inheritance of acquired characters. Did he imply that the "psychopathic constitution" inherited by this unfortunate family was first produced in an ancestor three hundred years ago, wrought somehow—by pangenesis, or other unexplained mechanism—into the structure of his germ-plasm, and so transmitted to his descendants? If yes, then I say this begs the whole question. If no, then the "psychopathic constitution" was not an acquired character, but a character already existing in that ancestor's germ-plasm. He transmitted the constitution he was born with; but that brings us no nearer an explanation of how he came to be born with it. But, incidentally, the stability of this dyscrasia in the germ-plasm for three hundred years on the whole confirms Weismann's doctrine of the continuity of the germ-plasm. Again, Dr Clouston strongly maintains that many of the insanities are manifestations of arrested development. But Weismann never denied the possibility of arrested development, which may arise from a

thousand causes other than inheritance of acquired characters. But Dr Clouston naturally asks why so many bad variations occur in the same family? This problem of the occurrence of variations that have selective value is tackled by Weismann in his "Germinal Selection," and most of the difficulties seem to me better correlated by that formula than by the doctrine of use-inheritance. In any case, even if use-inheritance were accepted, how can the transmission of so useless a variation as insane neurosis be accounted for? Dr Clouston says he does not believe that the child of an alcoholic parent is ever born with a taste for whisky; but he may be born with a lack of inhibition. But such lack of inhibition may very well have existed in the parent's constitution before alcoholism asserted itself; and, in any case, the nutrition of the germ-cells may be interfered with by the alcohol circulating in the parent's body, and germinal decrepitude may thus be produced. It is even *possible*, theoretically, that the nerve-elements of the germ-plasm (the nerve-determinants) may be so affected by alcohol that they grow into imperfect neurons or nerve-cells. But here again it is a case not of the transmission of characters acquired by the father, but of the effects of alcohol imbibed by the germ-cell itself. The case is on the same footing as a case of syphilitic infection, with this difference, that the poison of syphilis is a specific infection. The results of infecting or poisoning the germ-plasm may be anything from a temporary "illness" of the germ-cell to complete disintegration and death. The poisoning of the germ-plasm, however, has nothing to do with the transmission of a modification acquired by the parent. Other illustrations by Dr Clouston seemed equally explicable from Weismann's standpoint. He produced no case that contradicts the continuity of the germ-plasm or necessarily involves the inheritance of acquired characters. On the contrary, his cases are more simply explained without that assumption.

Dr James' precise position I was unable to grasp. I gather that he asserted the inheritance of acquired characters as too obvious to need discussion. But his illustrations—alcohol, phthisis and epilepsy—did not bear out his argument. Professor Hamilton's suggestion that these—and others—are all parts of the same constitutional dyscrasia seems to be admitted by Dr James. Why he should burden himself with the obscure and extravagant hypothesis of use-inheritance, I do not understand. Dr James says medical men have much more opportunity than biologists of knowing whether acquired characters are inherited. Possibly, but the characters are usually too complex for analysis. If they are inherited to the extent he assumes, cases ought to exist by the hundred thousand. It is somewhat disappointing that, with the single excep-

tion of Brown-Sequard's rough and inconclusive experiments on guinea-pigs and Dr Ballantyne's illustration of a possible case in pigeons, not one clear and criticised example of use or mutilation-inheritance was brought forward by any speaker. There are many examples much more plausible than Dr James'. The only definite biological facts produced (by Professor Ewart) told quite distinctly against such inheritance. As to Dr James' logical canon that what contradicts "common sense," and "common instinct" has something wrong with it, one is provoked to ask, with Cyrano, "*Que diable allait-il faire en cette galère?*" The discovery of the barometer, the application of the laws of gravity to the solar system, the doctrine Galileo was tortured for, the conservation of energy, natural selection, and many other great inductions, contradict common sense, which plays a very small part in the solution of any difficult and delicate problem.

Dr Ballantyne's remarks on "maternal impressions" are valuable, because he has pretty well done for that department of uncritical belief what Weismann did for the alleged inheritance of mutilations. Yet the so-called evidence for such impressions seems to me frequently stronger than the evidence for inheritance of acquired characters. When, however, Dr Ballantyne suggested antenatal mutilation for experiment, as more hopeful, he gave the general case away; for the acquired characters alleged to be transmitted are not the simple conditions induced by the very primitive environment of the uterine fluids, but the immensely delicate and complex integrations, dexterities, ideas, etc., developed, or *de novo* fashioned, in response to the complex environment of actual life. The case of Montaigne's calculus, which supervened at the same age as his father's, is an excellent example of the appearance of a germinal peculiarity in two branches of the same tree. Professor Schäfer's very important remark should give the "medical transmissionists" pause: infections are, by hypothesis, ruled out, and of the ultimate causation of other diseases we know little or nothing. For practical ends, pathology is an organised department; but biologically, disease is a *plus* or *minus* variation or modification, sometimes having selective value, sometimes not. Personally, I should be glad to believe, with Dr James, that much of these refinements in distinction are merely verbal, were I not persuaded by daily experience that the nightmare of the specific inheritance of acquired diseases overloads the spontaneity of life, paralyses the will, and hampers the preventive service in its efforts to improve the environment. Weismannism exalts the social inheritances, which, as the great organs of selection, constitute the basis of preventive medicine. But there are no data for a dogmatic conclusion on the modes of hereditary transmission of acquired or non-acquired characters.

Remarks by Dr GRAHAM BROWN.—It appears to me that the discussion turns very largely on a play on words. What do we mean by acquired characteristics? Do we mean such as were not present in the ancestry of the person in question? If we do mean some abnormality or divergency from the normal type, some alteration of tissue which may perhaps only show itself by an increased susceptibility to the action of external agency, then I am firmly of the belief that such acquired characteristics may be and often are handed down from father to son for generations.

Take, for example, the case of rheumatism. No one who has had any opportunity of studying this disease, and of acquiring a knowledge of the facts concerning it, can deny that three points at least are clear and well established.

1. That it prevails very much more in some families than in others.

2. That there is no evidence to show that it passes from person to person by infection or contagion.

3. That it is of the nature of an infective complaint.

Taking these points as admitted, it follows, to my thinking, that the occurrence of rheumatism in certain families can only be due to the transmission of some peculiar susceptibility of tissue from father to son.

Or take the case of alcoholism. As Dr Clouston has so admirably pointed out, an increased susceptibility to the action of alcohol (apart altogether from the alcohol craving) is noticed in neurotic families. This unusual vulnerability of the tissues to the action of alcohol can only, so far as I can see, be an acquired characteristic. I have certainly seen it in process of being acquired. That is, I have seen men who at first were able with impunity to take a fair quantity of alcohol who subsequently became alcoholic, and who having broken off the habit, found that in future even small quantities were apt to produce disagreeable results. The onus of proof lies on those who hold that these two forms of transmitted susceptibility of tissue are not acquired characteristics.

Remarks by Dr WILLIAM RUSSELL.—In the first place, I wish to express the pleasure it is to me and to many others here to see Professor Hamilton amongst us again. In listening to the discussion it has appeared to me that we have suffered from a want of definition in the terms used, and also a want of concentration on what seems to me to be the real point implied in the title of the opening paper. The existence of heredity is not questioned, but we have a biologist like Professor Cossar Ewart speaking as if nothing were to be regarded as heredity, unless

you could count it or figure it as number or arrangement of skin markings, the colour of feathers, or the number of them in a pigeon's tail. We are all interested in Professor Ewart's experiments, but most of them have no special bearing upon the question under discussion. The heredity which manifests itself in cross-breeding has only a general bearing on the influence of heredity on disease. We all know of curious hereditary anatomical characters in families: I know a family where webbing of fingers or toes prevails; but all that class of thing is not disease any more than are the colour of the eyes or the hair, or the size of the hands or feet. Another biologist tells us that we do not know what disease is; this is a strange statement, for those of us who deal with disease would say that in diseases not belonging to the infective series it begins as disturbed function, and the limit at which that disturbance begins is determined by the vigour of the cells of the part or organ affected. In the common matter of gastric digestion this is true; it is equally true of heart muscle. This is practically the same point as Dr Clouston so ably elaborated; it is the attainment of a "maturity" which is on a level at least with the average. This maturity is functional vigour. And will the biologist tell us that he knows an anatomical distinction which will enable us to say that one stomach is $x-$ and another $x+$? And because he cannot do this are we, as physicians and pathologists, to ignore this whole realm of observation as if it were a chimera, a mere dream? If biology is not prepared to take this teaching from us, it is doubtful what practical value biology can be of to us. I unhesitatingly range myself alongside Dr Clouston and say that the future of medicine depends more on this class of observation than on variations in pigeons' tails. The question before us seems to me to be this: (1) Can the chemico-vital activity and vigour of cells be influenced by the environment of the individual; can it be lowered, or modified, or altered? (2) Does that lowering or modification in any way influence the offspring along the same lines? There is surely only a positive answer to these two questions. Doubtless this modification may take generations to become fixed, for fortunately the *vis naturæ* tends to strengthen the weakened and to correct what has been modified, if the conditions are rendered more appropriate, more physiological, more hygienic; call it by what name we like, it means a favourable environment, and includes, as Dr Clouston has said, climate, food and drink. The variations and subtleties of the chemico-vital composition of individuals is vulgar knowledge to those of us who deal with deviations from the normal in living human beings. Take some examples of this. Some people are phenomenally vulnerable or susceptible to infective influences, while others are the reverse.

I knew a surgeon who could not make a *post-mortem* examination, or operate on a septic case, without getting poisoned. Many men work for years in the *post-mortem* room, and handle all things freely and are never poisoned. I know a medical man who took infective diseases whenever they became prevalent. He had, I believe, scarlet fever and typhoid several times; other medical men never have had either. Take what we call idiosyncrasy, even in the matter of food. I know a leading physician to whom a certain white fish is an emetic; to an old gentleman whom I have seen professionally, egg, in the smallest quantity and in any form, has always acted as an irritant poison, producing vomiting and diarrhœa; in another case a couple of strawberries produces a general urticaria. Passing from food to drugs, I once made a man as red as a lobster from head to feet by a small hypodermic of morphia. Within the last year I produced in an adult male private patient severe diarrhœa by giving him one or two one-drachm doses of the camphor water of the pharmacopœia. I presume we all meet these curious phenomena. Then if we turn to dyscrasia, are there no rheumatic and gouty people? Do we not recognise that these people have a modified vital chemistry which, under conditions to us harmless, manifests itself by what we clinically know by these names. In all these regions we are face to face constantly with what Professor Ewart would call an "epidemic of variation," not of coarse and palpable anatomical change, but of chemico-vital composition, which neither the biologist nor the physiological chemist can express even in symbols. Because they cannot do this, are we to regard this whole aspect of life as unreal, and existing only in our medical imaginings? I do not think we can too strongly repudiate any such suggestion.

There is certainly variation enough; how much of it is inherited, how much due to blends between male and female not physiologically successful, and how much acquired, are questions we cannot always answer. That acquired characters can be propagated there is strong proof. It is generally accepted that the two commonest infective diseases in this country, namely scarlet fever and measles, are much less virulent than they used to be, and this is almost certainly to be attributed, not to an attenuation of the virus, or to improved treatment, but to a measure of immunity acquired by a population whose progenitors for generations have passed through the ordeal of these infections. It is a historical fact that measles introduced into a virgin soil, unmodified by a degree of immunity acquired by transmission, proves a most virulent and fatal malady. Another historical instance is that of the company of Esquimaux who, taken to Berlin, were vaccinated to prevent them taking small-pox, and they nearly all died from the modified virus, used without any risk for protective purposes by the ordinary European.

We can now pass to Professor Hamilton's proposition that there is a hereditary anatomical type prone to certain definite diseases, and he illustrated this by reference to the general body type and the chest forms in persons liable to pulmonary tuberculosis, and which are well known. But this type and form constitute but a very small proportion of cases of pulmonary tuberculosis; and even where it does, the question is: Does not acquired tubercle determine the anatomical type in the offspring rather than the other way about? Even granting that the individual factor which allows entrance to the tubercle bacillus be an epithelium of low resisting power in the respiratory tract, is it for a moment to be allowed that that enfeeblement cannot be induced on the one hand, or strengthened on the other hand, by environment? Every physician says "No" to this, and the facts are so conspicuous that the matter need not be argued. While this enfeeblement of epithelium may be postulated and accepted as a working hypothesis, let us be perfectly clear in our own minds that this is not an anatomical distinction. No biologist or pathologist can say of a bronchial or alveolar epithelium that it has anatomical characters which distinguish it from another. We cannot move without postulating chemico-vital modifications and modifications of metabolic vigour which determine the beginnings of disease. I wholly believe with Dr Clouston that these modifications are acquired and transmitted. At an institution in this city I see a great many habitual alcoholics, and our inquiries show that in the greater proportion of them there is a marked family history; but the cases I see differ from those which come into Dr Clouston's experience, for comparatively few of them belong to the neurotic type. In my experience there is a periodic craving which the victims will do almost anything to satisfy, and which is a pathological condition only stateable as a chemico-vital perturbation, probably in nerve endings, which is transformed into conscious craving in cerebral cells. This tendency must be acquired, unless it is to be traced back to Noah; and even were this possible, the hereditary modification is not present in all his descendants, and must therefore have been eliminated in some by evolution. This morbid reaction to alcohol can, it seems to me, be, without any question, acquired, and it is as certainly transmissible. The same line of observation and of reasoning can be followed in rheumatism and gout —a vicious chemico-vital variation is transmitted, and most of us, I think, assume that the acquirement of this variation need not be thrown many generations back. The transmitted variation determines a great variety of anatomical change and of clinical phenomena, but the vitiated chemistry is none the less a real inheritance, because the coarse anatomical changes are not congenital, but supervene in adult life. To me the whole

question of the influence of heredity in disease centres in and radiates from this point of acquired and transmitted chemico-vital variations in cells, tissues, and organs.

Meeting VII.—May 2nd, 1900

MR A. GORDON MILLER, *President, in the Chair*

I. EXHIBITION OF PATIENT

Mr J. M. Cotterill exhibited a patient after LAMINECTOMY FOR PARALYSIS of two years' standing, the result of spinal caries. The symptoms of spinal mischief began when the patient was æt. 36, and signs of pressure on the cord developed rapidly. For six months before the operation there had been complete motor and sensory paralysis of the lower half of the body. There was incontinence of urine and fæces, and two large bed-sores had appeared on the buttocks. There was also cystitis. As counter-extension had given little relief, operation was decided upon, and the laminæ and spines of the eighth, ninth, and tenth dorsal vertebræ were removed. It was then found that pressure was due, not to granulation tissue, as was usually stated to be the case, but to exostoses springing from the bodies and arches of the vertebræ, which narrowed the spinal canal down to the diameter of a large quill. Improvement was manifested sixteen days after the operation, and the patient progressed steadily until he could now (a year after the operation) walk about with the aid of two sticks, and had complete control of his bladder and bowel.

II. EXHIBITION OF SPECIMENS

Mr J. Shaw M'Laren exhibited (*a*) a specimen of CARCINOMA OF THE ŒSOPHAGUS, and the stomach from the same case after gastrotomy had been performed. The patient had recovered and survived the operation some months. Considerable leakage had occurred for a week or two, though Witzel's operation had been performed. Mr M'Laren had not met with this before, but it was accounted for by the fact revealed at the *sectio* that the attempt to form an "artificial œsophagus" had failed in some way difficult to explain. The operation had been performed under eucain anæsthesia. The œsophagus was narrowed by the cancer, the tracheal mucous membrane

was ulcerated, and the growth had attached them, the aorta, and some glands and other blood-vessels in a large hard mass.

(*b*) A right and a left carpal scaphoid fractured into two almost equal portions by a fall from the roof of a house. It was by no means uncommon to find carpal bones broken in a case of a bad crush of the wrist, but a single fracture of one bone was very rare, while the occurrence of the fracture in each wrist added to the interest. The fracture was transverse in each case, and almost bisected the bone. There was no other fracture in the body. The patient died a few hours after the fall.

III. Original Communications

1. CROSS INFECTION, SO CALLED, IN FEVER HOSPITALS

By Claude B. Ker, M.D., M.R.C.P.Ed., Superintendent, City Fever Hospital, Edinburgh

Wherever large numbers of children are congregated together, whether in schools or in hospitals, there is always a risk that infectious disease may break out among them. It would seem, then, a perfectly logical deduction to make, that this risk will be materially increased if the place in which they are congregated happens to be a hospital in which infectious disease is treated. And so it is not surprising to find that many parents have a strong prejudice against sending their children into a fever hospital, lest they should, while resident there, contract another infection. And in this prejudice they are frequently encouraged by their family physician.

The hospital with which I am connected has, I am afraid, not a very good reputation as regards what is ordinarily known as cross infection, and I propose to consider how far that reputation is or is not deserved, and at the same time to remove some misapprehensions which apparently exist as to the nature and causes of this not infrequent occurrence. By cross infection is usually understood the contraction by the patient of some infection, other than that for which he was originally admitted. And it is always presumed that the second infection is derived, directly or indirectly, from patients who are being treated for that infection in some other part of the hospital.

Last year the City Hospital suffered rather more than usual as regards cross infection. I have tabulated the number of

patients treated for each disease, and the number who contracted a second infection, noting the percentage in each instance.

Of 1034 cases of scarlatina,	38, or 3·67	per cent.	took	measles.
" "	4, or ·38	"	"	varicella.
Of 331 cases of measles,	8, or 2·4	"	"	scarlatina.
Of 147 cases of diphtheria,	4, or 2·7	"	"	scarlatina.

Of these figures, I need only say that while the number of diphtherias which took scarlatina is about the normal, the other percentages are much higher than usual. It may also be noted that, of 472 patients admitted with infections other than the above, none took any other disease.

And now for the explanation of this state of affairs. The views most generally held by our critics outside may, I think, be fairly stated as follows:—(1) The second infection is contracted from patients suffering from that disease in other parts of the hospital. (2) That in many cases it is carried by the air. (3) That it is carried by the officials from one ward to another. (4) That the nurses mixing at meals contaminate each other, and so the patients under their charge. (5) That wards which have been used for the treatment of one disease are not properly disinfected before another disease is nursed in them.

It will be noticed that all these alleged causes depend on the assumption that the second infection is carried from patients in other parts of the hospital. While I am perfectly prepared to admit that such may be the case in certain rare instances, I assert that this method of infection is not at all a common one. For example, at the time measles first appeared in my scarlatina wards, the winter before last, not only were no cases of that disease being treated in the hospital, but there had not been a case in the ward where it broke out for over a year, although dozens of susceptible cases of scarlet had been treated there in the interim. Again, outbreaks of measles occur not infrequently in the magnificent hospitals of the Metropolitan Asylums Board, where measles cases are not treated at all.

As regards the carrying of infection by the air, as far as the City Hospital is concerned, it may be of interest to note that its measles wards are below those which are used for typhoid,

and may be almost said to ventilate into them by a staircase guarded only by a swing door. The scarlet wards are on the same flat as those for typhoid, and a similar swing door, used frequently by the medical and superintending staff, alone separates them.

I really hardly expect to be believed when I state the absolute fact that in the four years during which I have been in charge of the hospital not a single typhoid case, though at least 400 children must have been through the wards in that period, has developed either measles or scarlatina. Again, smallpox, that infection which of all others is most regarded as aerial, has before now been nursed in the narrow limits of our courtyard without accident. I have seen, on the other hand, two cases of true cross infection in the last four years, which were, I am convinced, due to aerial infection. Both were cases of measles, both were treated at different times in the same private ward, and both contracted scarlatina. The ward which they occupied is in an angle of the courtyard, and its window faces at the distance of a few feet a window of a scarlatina convalescent ward. When both windows were open it was easy to see that infection could be hardly avoided. I need not add that we are now careful whom we put in that private ward. But the case is altogether an exceptional one.

As regards the question of infection being carried by the staff, I quite admit that there must always be some slight risk of it. I have satisfied myself that a few cases of diphtheria have been so infected with scarlet, a disease to which they seem to be peculiarly susceptible. A doctor is very likely to be summoned in a great hurry to the diphtheria wards,—it may be from scarlet wards; an urgent message to see a diphtheritic croup may be the cause of some carelessness in personal disinfection. Latterly, my regulations for the protection of the diphtheria cases have been more stringent, and there has been no outbreak of scarlet which could not be more probably accounted for as being due to some more obvious cause.

That nurses occasionally infect each other in the dining-room is perfectly true, but I have no reason to believe that they have time to carry their disease to their patients before we isolate them. We propose in the new hospital, however, to make arrangements whereby the scarlatina nurses may be kept quite separate from the rest. The last cause alleged by

our critics is want of proper disinfection of wards. In my experience our disinfection is quite efficient, and any outbreak which may occur in wards, which have been used for another disease at some previous period, can always be accounted for in a much more obvious manner.

So much, then, for the alleged causes of the outbreaks which we are considering. For the real cause we have not to look far, it is imported infection. The most of our cross infection, in fact, is not cross infection at all; it does not come from other parts of the hospital, but from outside. And it may be introduced in a variety of ways.

First, it may be due to a wrong diagnosis made outside. I have often seen difficult cases of scarlet and diphtheria, or of scarlet and measles, confused. We, of course, do all we can to check the diagnosis on admission, but in many cases no signs of any disease at all are visible on admission, and if we did not accept the diagnosis, which is, of course, in the vast majority of cases perfectly correct, we would be compelled to isolate nearly a fourth of our admissions, which is manifestly impossible. For, even given the requisite number of isolation wards, it must be remembered that each case would require at least one, if not two, special nurses, that is, if the isolation is to be effectual. Occasionally, therefore, a case sent in as measles, and presenting no symptoms on admission, is found some days after to be desquamating, obviously from scarlatina, and it may or may not have infected others before we can get it removed.

Secondly, it may be due to a patient being admitted with two diseases, only one of which has been diagnosed. It is, I suppose, not necessary nowadays to point out, that two infections can exist simultaneously in the same patient. Not only is it possible, but it is comparatively common. Dr Johnston, in his report of the Glasgow Fever Hospitals for last year, notes no less than seventy-six cases admitted with two concurrent infections, and three in which three infections co-existed. In the last three months alone we have admitted more than a dozen. It will be readily seen how easy it is to miss one disease when another is quite obvious, and it may happen that attention is first drawn to the existence of the second infection by an outbreak of it in the other patients in the ward. It was in this manner that

scarlatina got among our measles and diphtheria patients last year.

Thirdly, and lastly, it is due to the admission of patients who are suffering from one disease and incubating another. The second infection, in the great majority of cases either measles or varicella, is developed some days after admission. This is by far the most common of the causes of so-called cross infection, and it is quite impossible to avoid. For the period of incubation presents no symptoms, and diseases which are infectious in their early stages only require a few hours in which to infect a whole ward. The outbreaks of measles among the scarlet cases in the City Hospital last year were due in both instances to cases of this kind. Dr Johnston, in the interesting report above alluded to, notes twenty-seven cases of this sort were admitted into the two Glasgow hospitals last year, and naturally caused infection in others.

It will be seen, then, from the above, that most of our second infections can be traced to the causes which I have given, and not to those which seem to be commonly supposed to be responsible for such outbreaks. And therefore it is clear that we will continue to have trouble of this sort, even after we occupy the new hospital which is in course of construction. The number of outbreaks will remain proportionately the same, but, owing to the fact that each individual ward can be completely isolated, the number of patients exposed in each case will be very considerably less. I hope, therefore, that there will be some diminution in the number of persons affected, but it is as well to recognise that no fever hospital, however modern, however perfectly administered, can expect immunity from these distressing accidents.

2. THE ACTIVITY OF THE SALIVA IN DISEASED CONDITIONS OF THE BODY.

By W. G. AITCHISON ROBERTSON, M.D., D.Sc., F.R.C.P.Ed., F.R.S.E., Lecturer on Medical Jurisprudence and Public Health, Edinburgh

BEFORE beginning the subject of this paper, I desire to point out a fact which is too often overlooked and which has been erroneously taught in the past. This is the all-important and primary action of saliva, that of transforming starch into maltose. Many teachers have been in the habit of stating that the amylolytic power of saliva is feeble and that any slight

transforming action which it possesses is soon brought to an end by reason of the acidity of the gastric secretion. Now it cannot be sufficiently inculcated that the converting power of ptyalin is extremely active and transforms a relatively enormous proportion of starch into sugar and this within a very brief period. It has been calculated that 120 grains of ptyalin are secreted each day as compared with 40 grains of pancreatin. When we think of the immense quantity of starchy food taken into the stomach each day, is it likely that Nature intended that this large amount of so active a ferment as ptyalin should be lost? It has been shown over and over again (as by von den Velden, Ewald, Sohlern, and others) that no free acid appears in the stomach until, at earliest, three-quarters of an hour after a mixed diet has been eaten. Consequently during this period the salivary ferment can exert its uncontrolled influence on starch in the stomach.

Not only so, but saliva has an alkaline reaction due most probably to the presence of alkaline phosphates and bicarbonates, while ptyalin acts most energetically in a neutral medium (neutralised saliva is more active than normal saliva—Chittenden and Ely). The neutralisation of the alkalinity of the saliva must therefore hinder the production of free acid. It has been also proved that the presence of a minute trace of free acid still further increases the activity of ptyalin. During the first period of gastric digestion the only free acids which may be produced are lactic or acetic, and these have a much feebler inhibitory effect on salivary proteolysis than hydrochloric acid. It is evident therefore that amylaceous matters must undergo under normal conditions a very thorough digestion in the stomach.

The salivary ferment is, however, destroyed by the gastric juice during the period of full proteid digestion. Thus only the more resistant portions of starchy food as, for example, lumps of imperfectly masticated potato, undercooked vegetables, rice, sago, tapioca, etc., may require a further disintegration and solution than they receive in the stomach. This they will obtain when they pass into the small intestine and come under the action of the amylopsin of the pancreatic secretion.

These facts dispose of the too prevalent idea that salivary digestion takes places only in the mouth and for a few minutes afterwards in the stomach.

Sécretion of Saliva.—It has been demonstrated by several observers, as by Hofbauer, that there is a daily variation in the diastatic action of saliva. Thus, on getting up in the morning the converting power of saliva is strong. It then falls towards and after breakfast, then rises slowly and reaches the maximum before the midday meal. After this it again falls rapidly and remains low for two hours subsequently. Towards the evening meal it rises slowly and this is succeeded by a gradual fall. So as to eliminate this variation in my experiments, the saliva was always collected at the same period during the evening.

Method of Investigation.—The experiments were performed in the following manner. Each individual was instructed to wash out his mouth thoroughly with slightly warm water and during the succeeding half-hour all the saliva which he secreted was received into a vessel and its quantity measured. Two cubic centimetres of the saliva were then mixed with ten cubic centimetres of starch mucilage at a temperature of 38° C. and the mixture was then kept at this temperature for ten minutes. At the end of this period the condition of the starch present was noted and further action of the ferment was prevented by rapidly boiling the mixture. The amount of sugar which had been formed by the ptyalin was then estimated by titration against standard Fehling's solution.

Healthy Adults.—The activity of the salivary ferment was investigated in ten healthy adults. If we take 0·080 gramme of sugar produced by the action of 2 c.c. saliva on 10 c.c. starch solution for ten minutes as a low average and as a means of estimating the intensity of salivary proteolytic action, we find that in healthy adults the amount of sugar produced has only in a single instance been below this figure. In almost every instance the amount of sugar produced has reached about 0·1 gramme and in some cases this figure has been greatly exceeded.

Children.—Under the age of five years the average amount of sugar formed is 0·077 gramme; from the ages of five to thirteen it is 0·078 gramme. The average figure all over from the ages of sixteen months to thirteen years is 0·078, the difference between the highest and lowest amounts being only 0·010 gramme. During childhood the activity of the salivary ferment remains remarkably uniform.

In Diseased Conditions.—Above one hundred cases of disease of various kinds were investigated in order to see if the activity

of the salivary ferment was altered. A glance over the figures shows us that the amount of sugar formed in such cases varied from 0 to 0·128 gramme.

If we analyse the table a little more closely we find that in the group

(1) *Gastro-intestinal disorders*, twenty-one cases were investigated. The average amount of sugar formed in these was 0·089 gramme. In chronic gastric catarrh this figure varied from 0·078 to 0·1 gramme. In acid dyspepsia the amount of sugar formed is above the healthy average. In ulceration of the stomach the amount of sugar formed is generally only slightly below the normal average. These general observations agree in the main with the conclusions arrived at by two Italian investigators, Petterutti and Ferro, who state that the greater number of gastro-intestinal disorders are associated with a decrease in the amount of ptyalin.

In one case of great dilatation of the stomach the salivary ferment proved to be almost absent or at least inactive in the saliva.

In cirrhosis of the liver the amount of sugar is by no means reduced, and even in some cases it is greatly increased.

(2) *Pulmonary Diseases.*—In the group of lung diseases generally the salivary ferment is fairly active, and on an average 0·087 gramme of sugar was formed. In the cases of phthisis which were examined ptyalin was present in normal amount, as the sugar formed reached almost always to the standard quantity. The cases of pneumonia which I examined furnished results similar to those obtained by Jawein. These cases showed that the amylolytic action of the saliva is higher than the normal during the period preceding the crisis, but lower after the crisis.

(3) *Heart Diseases.*—In the large group of heart cases the saliva retains its usual composition, and the amount of sugar formed in my experiments hovered at or about the normal limits.

(4) *Nervous Diseases.*—A larger proportion of subnormal cases were met with in the group of nervous diseases (41 per cent. giving a proportion of sugar lower than the normal average). In one case of cerebral tumour the saliva, though copious in amount, contained practically no converting ferment, whereas in a case of locomotor ataxia, though the secretion was equally copious, the converting ferment produced the large amount of 0·111 gramme sugar.

(5) *Hæmopoietic System.*—Of three cases of Addison's disease examined, the saliva of two showed marked deficiency in its power of starch digestion, while the third exceeded the normal limit.

(6) *Renal Diseases.*—Taken all over, the group of renal diseases shows a lower average than the normal. In 55·5 per cent. the quantity of sugar formed was considerably below the average.

In diabetes, on the other hand, the saliva has a very active converting action. In three out of four cases examined the average proportion of sugar formed was much above the standard figure. The saliva in these cases contained no traces of sugar.

In simple anæmia the converting ferment seems to be present in the saliva in its normal amount. If, however, the anæmia be associated with dyspepsia, the average is subnormal.

In sub-acute and chronic rheumatism the ferment shows no great variations from the normal.

In general febrile conditions the secretion of saliva is greatly reduced in amount, and this reduction increases *pari passu* with the increase in temperature. It is important to note that this scanty secretion seems to be possessed of enhanced amylolytic power. This same fact has also been noted by Jawein.

Quantity of Saliva Secreted.—If we pass now to the consideration of the amount of saliva secreted in the various diseased conditions of the body, and taking as the normal amount secreted by healthy adults as from 10 to 15 c.c. in half an hour, we note first that in infants and children the amount of saliva poured out is relatively much greater than it is in adults. For example, in five children under the ages of three years, and who could not be made to understand properly how to collect their saliva, from 7 to 9 c.c. were obtained in thirty minutes. In two girls aged four years the amounts were respectively 12 and 14 c.c. In another girl aged five 20 c.c. were secreted, and so on. In the case of females the secretion seems to be generally a little more copious than in males.

Gastric Disorders.—In most cases of Acid Dyspepsia the secretion is above the normal in amount. This may be due to reflex irritation of the salivary glands. One authority has recommended patients suffering from this affection to further stimulate the secretion of saliva by sucking gelatine lozenges.

He thinks that the large amount of alkaline saliva thus introduced into the stomach will neutralise to a great extent the hyperacidity of the gastric secretion.

In Chronic Gastric Catarrh the amount secreted is hardly, as a rule, up to the average. In Ulceration of the Stomach the same deficiency of secretion is met with.

In those cases where we find Diarrhœa or Ascites the secretion of saliva is in many cases far below the normal amount.

Pulmonary Diseases.—In the group of diseases of the lungs we find that in Bronchitis and in the early stage of Pneumonia the volume of saliva which is secreted is generally up to the full average, and may even be beyond it. In the case of Chronic Phthisis, on the other hand, the secretion of saliva is always very scanty.

Cardiac Diseases.—In the large class of heart affections of a grave nature there is an almost constant diminution in the secretion.

Nervous Diseases.—In affections of the cord, the amount of saliva secreted reaches the highest average, and even surpasses it, as, for example, locomotor ataxia. In hemiplegia the same excess is found. The reverse is found generally in the class of cerebral tumours where the saliva is very small in amount.

Renal Diseases.—In chronic Bright's disease as a rule we see that the salivary secretion is scanty. This may account for the very fœtid odour of the breath so often present in such cases. In diabetes, of course, the saliva is very scantily secreted.

In *Simple Anæmia*, in the *Chronic forms of Rheumatism*, and in three cases of *Addison's disease* examined, the amount of saliva was sub-normal.

In *Fevers* generally, when the temperature is at all high, the secretion of saliva is lessened in amount, but (as has already been noted) its amylolytic power is increased. In very slight degrees of fever, however, the normal amount may be produced.

It might be thought that where a large amount of liquid saliva is secreted the ferment would not be in so large an amount as it would be in an equal volume of saliva where its secretion was scanty. Such an opinion, though true in the main, is not in all cases borne out by a consideration of the

facts observed, either in the healthy or diseased conditions. In many cases where the secretion is scanty, the amount of ptyalin is also small; while in other cases where the secretion is copious, its proteolytic power is also great.

It is an easy matter for anyone to satisfy himself at once by a simple examination of the saliva as to whether unchanged starchy food ought to be given to an invalid or not. Of course, if the secretion be very scanty, as, for example, in pyrexia, then it would be quite out of the question to think of administering such a food, and practically we know that such fever cases are usually nourished on milk, meat infusions or jellies, alcohol or pre-digested starchy foods.

If, on the other hand, the secretion be present, but there is doubt as to its amylolytic power, then the experiment is readily performed. A small quantity (about 10 c.c.) of starch mucilage is to be mixed with 1 or 2 c.c. of the saliva in a test tube, and the whole is to be kept at a temperature of 100° F. for a few minutes (five to ten). This can readily be accomplished by immersing the tube in a large body of warm water, and taking the temperature of this by means of a clinical thermometer. At the end of this period the mixture is to be boiled. If a few drops of this be now poured on to a plate and a drop of a weak solution of iodine be added, the degree of starch conversion will be at once shown. If the ptyalin has had no action, then the starch is merely coloured blue or violet, but if the ferment has been active, then a reddish-purple or red-pink colour is produced, depending on the intensity of the action. If the ferment has been energetic no colour is produced, the whole of the starch having been converted into the colourless form of dextrin (achroodextrin). The latter result is seldom obtained, and we generally find a more or less red-purple colour, indicating that only part of the starch has undergone conversion. The redder or pinker the colour the greater has been the power of the ptyalin.

Next, a small quantity of Fehling's solution is boiled in a test tube, and one drop of the mixture is added. If no precipitation of the suboxide of copper takes place a second drop is added, and so on until a precipitate is produced. Such a proceeding gives a very rough estimate of the amount of maltose produced, and consequently of the intensity of the salivary ferment.

These two tests are so simple and easily performed that no one need neglect the examination of the saliva in a doubtful case.

It is evident that a full appreciation of the amylolytic power of saliva is of immense value when we consider the dietetics of disease. In those cases where the salivary secretion is extremely scanty or even in abeyance, as, for example, general pyrexia, the exanthemata, etc., or in such cases as dilatation of the stomach, where the saliva contains almost no ptyalin, or again in cases where the secretion has been inhibited by drugs (*e.g.* morphin, atropin) it is evident that ordinary starchy foods ought not to be allowed. If they are administered at all, the starch must be in the first place rendered soluble by treatment with some of the artificial digestant agents as extract of malt, zymine, taka-diastase, etc. These facts are of course insisted on in every reliable work on dietetics, but in spite of this, they are generally ignored in practice. One authority, for example, recommends the giving of farinaceous foods in dilatation of the stomach, and in this disease we have seen that the proteolytic action of the saliva is very feeble or even absent.

In conclusion, I would advocate a systematic examination of the saliva in all diseases of the gastro-intestinal tract as also in other diseases where ordinary starchy food is found to disagree with the patient.

AMOUNT AND CONVERTING POWER OF SALIVA.
HEALTHY ADULTS.

No.	Sex.	Age.	No. c.c. secreted.	2 c.c. saliva and 10 c.c. starch sol. at 38 C. for 10 minutes.	
				Changes in starch.	Amount of sugar formed.
1	Male	34 yrs.	12	Sol. starch much, erythro. and achro. some	·076
2	"	20 "	10	" "	·094
3	"	22 "	12	" "	·094
4	"	34 "	16	" "	·121
5	"	21 "	5	" "	·092
6	"	19 "	5	" "	·1
7	"	22 "	3	" "	·113
8	"	20 "	4	" "	·080
9	Female	32 "	16	" "	·067

HEALTHY CHILDREN.

No.	Sex.	Age.	No. c.c. secreted.	2 c.c. saliva and 10 c.c. starch sol. at 38 C. for 10 minutes.	
				Changes in starch.	Amount of sugar formed.
1	Female	16 mo.	7	Sol. eryth. achroo.	·081
2	"	17 "	7	" "	·073
3	Male	19 "	9	" "	·078
4	Female	2 yrs.	8	" "	·079
5	Male	3 "	8	" "	·075
6	Female	4 "	12	" "	·080
7	"	4 "	14	" "	·075
8	"	5 "	20	" "	·078
9	"	7 "	12	" "	·080
10	Male	7 "	12	" "	·073
11	Female	8 "	7	" "	·083
12	"	10 "	8	" "	·080
13	Male	12 "	16	" "	·081
14	Female	12 "	12	" "	·080
15	"	13 "	14	" "	·075

DISEASED CONDITIONS.

No.	Disease.	Sex.	Age.	No. c.c. sec.	2 c.c. saliva and 10 c.c. starch sol. at 38 C.	
					Changes in starch.	Amount of sugar formed.
1	Acid dyspepsia	F	50	17 acid react.	Soluble starch much, erythro. some	0·108 gr.
2	Acid dyspepsia	M	22	4	Sol. starch only	0·106
3	Chronic gastric catarrh	M	71	8	Sol. and eryth. much	0·078
4	Do. do.	M	47	10	Do. do.	0·086
5	Do. do.	M	44	9	Much sol., some eryth.	0·087
6	Do., with fermentation	M	40	4	Much sol., little eryth.	0·10
7	Dilatation of stomach (great)	M	45	8	Sol. starch only	0·008
8	Gastric ulcer with hæmoptysis	M	27	10	Sol. and eryth.	0·079
9	Do. do.	F	30	6	Sol. much	0·078
10	Simple gastric ulcer with anæmia	F	17	10	Sol. much	0·073
11	Gastric ulcer	F	23	9	Sol. much	0·083
12	Diarrhœa (nervous)	M	33	6	Sol. and eryth.	0·073
13	Tubercular disease of the bowels	M	13	0	Mouth perfectly dry	...
14	Peritonitis	F	20	11	Sol. and eryth.	0·113
15	Ascites from cancer of liver	F	42	10	Sol. much, eryth. little	0·086
16	Ascites	M	39	2	Sol., eryth., achro.	0·113

DISEASED CONDITIONS—*continued.*

No.	Disease.	Sex.	Age.	No. c.c. sec.	2 c.c. saliva and 10 c.c. starch sol. at 38 c.	
					Changes in starch.	Amount of sugar formed.
17	Ascites from cirrhosis of liver	M	63	8	Soluble, erythro. achro.	0·108 gr.
18	Do. do.	M	34	3	Do. do.	0·080
19	Ascites	M	38	3	Do. do.	0·113
20	Ascites with cirrhotic liver	M	36	14	Do. do.	0·080
21	Do. do.	M	40	10	Do. do.	0·096
22	Gallstones	M	30	8	Do. do.	0·113
23	Bronchitis	M	44	9	Much sol. and erythro.	0·092
24	Do. with some pneumonia	M	24	21	Do. do.	0·094
25	Pleurisy and bronchitis	M	26	20	Do. do.	0·084
26	Pneumonia	M	24	21	Do. do.	0·104
27	Do. after crisis	M	48	6	Do. do.	0·081
28	Phthisis	M	41	4	Much sol., little erythro. or achro.	0·074
29	Do.	M	20	12	Do. do.	0·098
30	Do.	M	49	2	Do. do.	0·104
31	Do.	F	21	8	Do. do.	0·067
32	Do.	M	36	2	Do. do.	0·080
33	Cardiac weakness	M	54	4	Sol., erythro. and achro.	0·092
34	Cardiac weakness—dropsy	M	59	2	Do. do.	0·086
35	Mitral heart disease	M	51	4	Do. do.	0·102
36	Mitral heart disease (temp. 102° F.)	F	25	5	Sol., erythro. much, achro.	0·092
37	Mitral heart disease	M	18	26	Do. do.	0·064
38	Mitral heart disease (advanced)	M	26	5	Do. do.	0·080
39	Mitral heart disease	M	20	16	Sol. erythro, much	0·075
40	Double mitral disease with nephritis	M	30	6	Sol. much, erythro.	0·078
41	Mitral disease	F	56	5	Do. do.	0·083
42	Mitral stenosis	M	32	12	Do. do.	0·080
43	Do. with bronchitis	F	19	11	Do. do.	0·070
44	Double mitral and aortic disease—dropsy	M	20	6	Do. do.	0·003
45	Mitral heart disease—dropsy	M	39	12	Do. do.	0·086
46	Double aortic disease	M	41	21	Do. do.	0·080
47	Aortic aneurism	M	36	5	Do. do.	0·078
48	General debility	M	55	4	Do. do.	0·086
49	Chorea	F	12	8	Do. do.	0·080
50	Chorea (chronic)	M	20	6	Do. do.	0·075

DISEASED CONDITIONS—*continued.*

No.	Disease.	Sex.	Age.	No. c.c. sec.	2 c.c. saliva and 10 c.c. starch sol. at 38 C.	
					Changes in starch.	Amount of sugar formed.
51	Sciatica (severe)	M	44	5	Sol. much	0·062 gr.
52	Do. with great debility	M	62	7	Do. erythro.	0·083
53	Hysteria	F	18	5	Sol., erythro.	...
54	Neuritis, Myelitis	M	56	17	Do. do.	0·083
55	Progressive muscular atrophy	M	19	17	Sol. much, erythro.	0·080
56	Paraplegia	M	44	18	Do. do.	0·082
57	Do.	M	57	24	Sol. much	0·068
58	Spastic Paralysis	M	38	9	Sol. much, erythro.	0·075
59	Hemiplegia	M	40	17	Do. do.	0·083
60	Do.	M	26	15	Do. do.	0·073
61	Locomotor ataxia	M	32	22	Do. do.	0·111
62	Cerebral tumour	F	27	8	Do. do.	0·096
63	Do. do.	F	30	3	Do. do.	0·10
64	Do. do.	M	32	15	Sol. only.	0·001
65	Do. do.	F	19	12	Sol. much	0·084
66	Exophthalmic goitre	F	27	6	Do. do.	0.083
67	Purpura hæmorrhagia (Henoch's)	M	13	22	Sol, erythro., achro.	0·10
68	Addison's disease	M	17	11	Do. do.	0·104
69	Do. do.	M	30	12	Sol., erythro.	0·067
70	Do. do.	F	23	3	Do. do.	0·064
71	Solid œdema of face	M	28	12	Do. do.	0·078
72	Nephritis (acute)	M	50	14	Sol. much, erythro.	0·086
73	Do. do.	M	33	7	Sol., erythro.	...
74	Do. do.	M	21	4	Do.	0·089
75	Do. do.	M	8	12	Sol. much.	0·096
76	Do. do.	M	46	6	Sol., erythro.	0·069
77	Nephritis (chronic)	M	19	4	Sol. much.	0·083
78	Do. do.	F	24	10	Do.	0·066
79	Do. do.	F	64	2	Do.	0·076
80	Do. with bronchitis	M	64	14	Do.	0·078
81	Diabetes (Mellitus)	F	50	6	Sol., erythro., achro.	0·060
82	Do. do.	M	44	8	Do. do.	0·108
83	Do. do.	M	46	2	Do. do.	0·104
84	Do. do.	M	23	6	Sol. much.	0·104
85	Anæmia.	F	18	6	Sol., erythro.	0·083
86	Do.	F	17	4	Do.	0·089
87	Do. with dyspepsia	F	21	8	Sol. much.	0·081
88	Do. do.	F	18	5	Do.	0·069
89	Rheumatism.	F	33	2	Sol., erythro.	0·064
90	Do.	F	47	3	Do.	0·086
91	Rheumatic fever	F	15	7	Sol., erythro.	0·094
92	Rheumatic fever, subacute	F	27	6	Sol., erythro.	0·086
93	Do. do.	F	24	2	Do.	0·078
94	Do. do.	F	25	13	Do. achro.	0·089

DISEASED CONDITIONS—*continued.*

No.	Disease.	Sex.	Age.	No. c.c. sec.	2 c.c. saliva and 10 c.c. starch sol. at 38 c.	
					Changes in starch.	Amount of sugar formed.
95	Influenza	M	56	4	Sol., erythro., achro.	0·128 gr.
96	Do.	M	29	5	Sol. erythro.	0·098
97	Quinsy	F	18	4	Do. achro.	0·083
98	Suppurating hip joint	M	24	16	Do.	0·089
99	Chronic suppuration, waxy disease	M	21	6	Do.	0·075
100	Eczema, acute widespread	M	37	16	Do. do.	0·098
101	Acromegaly	F	33	12	Do. do.	0·102

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Meeting VIII.—June 13, 1900

Mr A. GORDON MILLER, *President, in the Chair*

I. ELECTION OF HONORARY MEMBERS

The following were elected honorary members of the Society:—Sir Thomas Lauder Brunton, M.D., LL.D., D.Sc., F.R.C.P.L., F.R.S., London; Professor William Osler, M.D., LL.D., F.R.S., F.R.C.P., Baltimore; Dr Hermann Snellen, Emeritus-Professor of Ophthalmology, Utrecht.

II. EXHIBITION OF PATIENTS

1. *Dr James Cameron* exhibited a case of ADIPOSIS DOLOROSA (Dercum). The patient is a woman of about 50 years of age. For the past twenty-five years she has been gradually increasing in obesity.

When seen at Christmas 1899, she was so stout that she was unable to get out of bed. The mammæ and labia were enormously developed. The specific fatty tumours in the shins were very well marked, while from the hypogastric region there hung a fatty tumour the size of a cocoa nut.

The hands and feet were not enlarged and the face was normal.

The pains were present and very severe, at times necessitating the use of anodyne applications.

There were no mental symptoms as is often the case in the disease.

The patient has improved very much under the administration of thyroid along with Fowler's Solution, and has recently been taking five 5-gr. tablets daily, so that she is now able to get about.

Quite recently Dercum found in a case examined post-mortem, that the thyroid was atrophied and the colloid material absent.

This may explain the improvement under thyroid treatment here, but apart from this Dr Cameron previously found it of benefit in a less aggravated case of obesity than the present example.

2. *Mr C. W. Cathcart* exhibited (*a*) a boy after laparotomy for INTESTINAL OBSTRUCTION. This case illustrated in an unusual way the advantage of emptying the bowel above the seat of obstruction in addition to making sure that the lumen of the gut has been re-established. The patient was admitted on January 24th to the Royal Infirmary late at night, three days after symptoms of obstruction had appeared. Mr Cathcart operated next day and found the bowel adherent to a caseous mesenteric gland and obstructed partly by being acutely bent on itself and partly by being surrounded with bands of lymph. After the obstruction was relieved the loaded bowel above was opened and emptied through a glass drainage tube temporarily tied in. This tube became blocked several times with masses of undigested and probably indigestible material—shown to the meeting—and had to be cleared with a long probe. The opening was immediately afterwards closed with Lembert sutures. These masses would probable have blocked the bowel again which was narrowed at the seat of

obstruction. For a few days the boy was in an anxious condition, but improved steadily after the bowels had been induced to act by the aid of calomel.

Previous to this attack the boy had been in delicate health —troubled with diarrhœa and constipation alternately, and liable to attacks of abdominal pain from chills or errors of diet. His symptoms began at night, and the day before some well intentioned friends had treated him to an Italian salad, after which he had eaten an apple and fried potatoes. This explains the masses which blocked the tube.

(*b*) A woman, aged , after excision of bowel for gangrene due to STRANGULATED FEMORAL HERNIA. When the patient was admitted to Ward 14 she had an inflamed and tender lump at the right saphenous opening and a history of abdominal pain and sickness for about four days. She had been previously examined for hernia, and only a small lump, like an enlarged gland, at the right saphenous opening had been found. This had not been recognised as a hernia. Her bowels, too, which had been constipated for two days before the other symptoms had began, had been well moved by an enema two days before the operation. However, the continuation of the abdominal symptoms and the appearance of redness and swelling in the crural region, showed the case, when admitted to Ward 14, to be one where there was probably gangrene supervening on strangulation. The operation, which was performed immediately, proved this to be the case. The sac was so soft and grey that it was at first taken to be gangrenous bowel. When opened the sac contained a portion only of the wall of the bowel (Richter's Hernia). This explains why the strangulated part was so small at first, the subsequent redness and swelling being due to inflammation following the gangrene.

Such cases are not only difficult at first to diagnose but are, when gangrenous, less amenable to the formation of an artificial anus, than when a knuckle of bowel is involved. As the patient's strength was good the adjacent bowel was drawn down and excised, the loaded portions above having been previously evacuated. The ends were united with a double row of continuous fine silk stitches. The wound was well swabbed with pure carbolic and stuffed with iodoform gauze. It healed by granulation and has thus produced a radical cure.

The patient was not very strong before the operation, but is now convalescing steadily and has no trouble with her bowels. The portion of bowel removed was shown.

III. Exhibition of Pathological Specimen

Mr C. W. Cathcart exhibited a portion of the SIGMOID FLEXURE, removed for a new growth which had caused obstruction.

The patient was a gentleman of about 83 years of age, who had suffered for some years from constipation with occasional passage of blood and mucus. In November 1899, about twelve hours before the operation, he had been seized with severe tenesmus but without any relief of the bowels. An intussusception presented at the anus. As this could not be reduced by enemata and pressure from below, the abdomen was opened in the left flank, the intussusception (formed by the sigmoid flexure), with difficulty reduced, and the new growth recognised about the middle of an unusually ample sigmoid flexure. The bowel was evacuated through a Paul's tube and the central part of the sigmoid flexure containing the tumour was excised. The cut ends were immediately re-united with a double row of silk stitches, and the patient has made a complete and satisfactory recovery.

IV. Exhibition of Instrument

Mr C. W. Cathcart exhibited a SURGICAL OPERATING CASE. The objects aimed at were to have a case which could be easily cleaned and in which the numerous articles required for a capital operation could be conveniently stowed away and easily unpacked. This has been carried out by a case of polished zinc, which has a deep pocket for bottles and a removable tray for smaller articles which are advantageously kept by themselves. The lid and one side are also removable and form convenient trays on which to lay out what is required for the operation. The instruments are carried in a metal box provided with a perforated false bottom. Each half of the box forms a tray. Before an operation the instruments are scalded in one of these by having boiling water poured over them. When it is desired to cool them they are transferred by means of the false bottom from the hot tray to the other one containing cold

lotion. After use the instruments are boiled at home. Some are stored in the metal box ready for use; the others are stored in dust proof jars, which are simply confection jars provided with a layer of wool or lint where the lid fits on to the jar. This is not only a much cheaper but a more efficient protection from dust than the elaborate glass and metal cases sold for the purpose, while the use of such jars facilitates the storing together of similar forms of instrument.

The large metal case is furnished with a water-proof canvas cover which has a large envelope pocket, occupying one side, for the accommodation of extras.

The maker is Mr H. Gilchrist, 22 Nicolson Street, who charges for the large case £1, 10s.; for the cover 18s. 6d.; for the larger boxes 16s. 6d., and for a smaller size 12s. 6d.

V. Original Communications

1. SOME OBSERVATIONS UPON THE GENERAL BLOOD PRESSURE IN SLEEPLESSNESS AND SLEEP

By Lewis C. Bruce, M.D., Physician Superintendent, Perth District Asylum, Murthly

The following observations made upon the general blood pressure in states of sleeplessness and sleep were made upon inmates of Murthly Asylum.

The observations made during conditions of sleeplessness were of necessity made upon patients who were suffering from acute mental disease, but I have only recorded cases in whom sleeplessness was an outstanding symptom and not just an accidental occurrence. The observations upon sleep were all obtained from convalescent patients who were sleeping well.

The instrument used was Barnard & Hill's sphygmometer, and I take for granted that Hill's statement that "the normal general arterial pressure in the horizontal posture equals about 110 mm. Hg." is correct.

The first points upon which I desired to obtain information were:—

1. Are there any unusual conditions of blood pressure which are associated with sleeplessness in mental disease?

2. What changes occur in the blood pressure when sleep is induced in these cases by drugs?

In all, sixteen acute, recently admitted, cases of mental disease with sleeplessness as a marked symptom gave reliable results.

The blood pressure in twelve of these cases with sleeplessness ranged from 130 to 150 mm. Hg., and in the remaining four cases the blood pressures ranged from 110 to 100 mm. Hg.

The cases, therefore, seemed to range themselves under two classes :—

1. Sleeplessness with high blood pressure.
2. Sleeplessness with normal or low blood pressure.

The drugs used to induce sleep in these patients were: paraldehyde, sulphonal, trional, and potassium bromide.

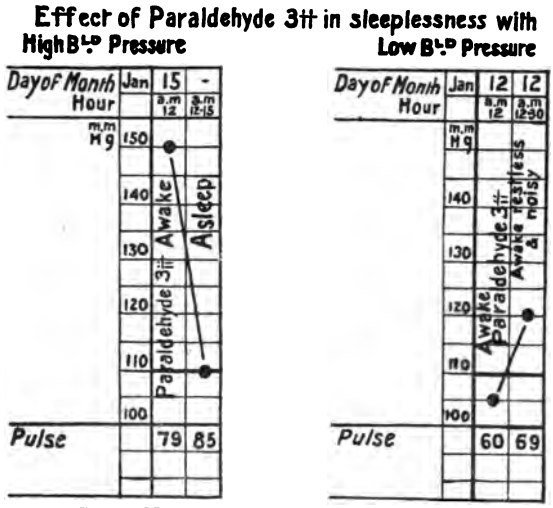


CHART No. 1. CHART No. 2.

Chart No. 1 shows the effect of paraldehyde in ℥ii doses upon a blood pressure of 150 mm. Hg. with sleeplessness. At 12 midnight the patient was sleepless, and the blood pressure stood at 150 mm. Hg. Paraldehyde ℥ii was administered. At 12.15 A.M., *i.e.* fifteen minutes later, the patient was asleep, and the blood pressure had fallen to 110 mm. Hg.

Chart No. 2.—Shows the effect of paraldehyde in ℥ii doses upon a blood pressure of 105 mm. Hg. with sleeplessness. At 12 midnight the patient was sleepless, with a blood pressure of 105 mm. Hg. Paraldehyde ℥ii was administered. Almost immediately the patient became talkative, restless, and at 12·30 A.M., half an hour later was still restless. The tension had risen to 120 mm. Hg., and the patient did no sleep until three hours later.

Paraldehyde given in large doses of ℥ii always induced sleep in class 1—sleeplessness with high blood pressure. Sleep followed as a rule within fifteen minutes after the administra-

tion of the drug, and the blood pressure fell either to 110 mm. Hg. or below 110 mm. Hg. (see Chart 1).

In class No. 2—sleeplessness with low blood pressure—paraldehyde in ʒii doses invariably failed to induce satisfactory sleep.

In these cases immediately after the administration of the drug the patients became restless, their blood pressures rose, and they either failed to sleep, or if sleep did ultimately set in, it was of a restless, muttering, unrefreshing character (see Chart No. 2). I ultimately found that in these same patients small doses of paraldehyde—15 to 30 mins.—generally induced sleep rapidly.

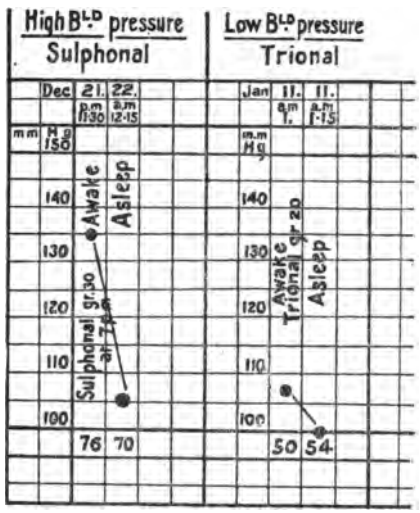


CHART No. 3. CHART No. 4.

Chart No. 3.—Shows the effect of sulphonal gr. 30 on a case of sleeplessness with a blood pressure of 135 mm. Hg. When sleep set in the blood pressure fell to 105 mm. Hg.

Chart No. 4.—Shows the effect of trional gr. 20 on a case of sleeplessness with a blood pressure of 105 mm. Hg. In fifteen minutes after the drug was administered the patient was asleep, and the blood pressure was 100 mm. Hg.

I class sulphonal and trional together, as they gave very similar results—with the difference that trional acted more rapidly than the sulphonal, and was less reliable in cases of sleeplessness with high blood pressure.

In class No. 1—sleeplessness with high blood pressure—they were not nearly such certain sleep producers as paraldehyde, and the resulting sleep did not seem to be so refreshing. When sleep did set in, however, the blood pressure always fell to 110 mm. Hg. or lower, and, upon the other hand, they never failed to produce sleep in class 2—sleeplessness with low blood pressure (see Charts 3 and 4).

I obtained no satisfactory readings with potassium bromide.

It would appear, then, that in the majority of cases of sleeplessness in mental disease the sleeplessness is associated with a high blood pressure, and that the most certain hypnotic is paraldehyde given in large doses of at least ʒii, and it is

also certain that when sleep is induced in these cases by paraldehyde, or any other drug, that the blood pressure falls to 110 mm. Hg. or below 110 mm. Hg.

There also appear to be a certain proportion of cases of sleeplessness with low blood pressure—of 110 or below 110 mm. Hg. This class is, I believe, best treated by sulphonal or trional, or small doses of paraldehyde. When sleep ensues in these cases as the result of drugs, the blood pressure does not necessarily undergo much change (see Chart 4).

As all the cases of sleeplessness with high blood pressure, when they slept under the influence of drugs, showed a fall in blood pressure, I determined to try the effect of using, as a hypnotic in these cases, a drug which had no other action than reducing blood pressure. Such a drug is erythrol tetra-nitrate. Up to the present I have applied this drug in nine cases of sleeplessness with high blood pressure. Of these nine cases, three were immediately and permanently benefited; three were temporarily improved for two or three nights, and then the drug appeared to lose its effect, their blood pressures rose, and sleeplessness again became a troublesome symptom. In the remaining three cases the drug had absolutely no effect. The patients upon whom the drug had no effect were two cases of acute mania and one case of senility. In all these cases ½ gr. of erythrol tetra-nitrate was administered night and morning. If the blood pressure was not reduced the dose was increased gradually to 2 grs. per diem, and if this amount failed to reduce the blood pressure and induce sleep, treatment was discontinued.

In my experience, erythrol tetra-nitrate is a drug that will act at once if it is going to act at all, and our three successful cases have so impressed me that I intend to give erythrol tetra-nitrate a trial as the first hypnotic in every case of sleeplessness associated with high blood pressure (see Charts 8A and 13).

In states of over-fatigue, especially mental over-fatigue, the blood appears always to be run at high pressure, and sleeplessness is one of the first symptoms, if not the first symptom which indicates that there is going to be a nervous breakdown. In many such early cases I believe erythrol tetra-nitrate might be found useful in producing sleep by reducing blood pressure.

I have used erythrol tetra-nitrate extensively in various

conditions of raised arterial pressure, and have never found it give rise to unpleasant symptoms, except in two patients who suffered from bad headache after taking ½ gr. doses of the drug. It is, however, said to be a dangerous drug, in that it is an explosive of a violent nature.[1]

The second series of observations were made upon con-

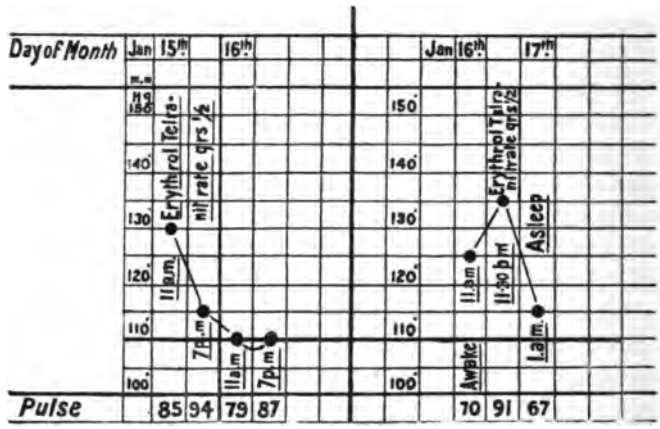


CHART No. 8A. CHART No. 13.

Chart No. 8a.—Shows the effect of 1 gr. of erythrol tetra-nitrate per diem upon a patient who was suffering from melancholia with sleeplessness and a high blood pressure. Up to the 15th of January this patient had not slept for fourteen days for more than an hour at a time. From the 15th January onwards she slept soundly and recovered rapidly.

Chart No. 13.—Shows the effect of erythrol tetra-nitrate gr. ½ as a sleep producer. This patient, also a case of melancholia, had not slept for two nights. At 11.30 P.M. erythrol tetra-nitrate gr. ½ was administered. In less than an hour she was asleep, and at 1 A.M. the pressure had fallen to 115 mm. Hg.

valescent patients in order to throw some light upon the following questions:—

1. Is sleep possible with blood pressures of 130 mm. Hg. or higher?

2. What relation does the morning blood pressure bear to the evening pressure when a person is sleeping well?

3. Are there any differences in the blood pressure of the same person asleep under the action of drugs and sleeping naturally?

[1] With the assistance of my colleague, Dr Alexander, I have tried to explode erythrol-tetra-nitrate by detonation. We failed to produce any explosion, and we are of the opinion that erythrol-tetra-nitrate as supplied in tabloids cannot be an easily exploded form of the drug.

(1) Is sleep possible with high blood pressure?

Among some thirty records of blood pressures taken from sleeping patients, I have two charts of natural sleep which show pressures of 130 mm. Hg. and 140 mm. Hg. during sleep. The explanation is, I believe, as follows:—Both these patients had just passed into the condition of sleep when the record was taken, for on taking the pressure again an hour later in the sleeper whose blood pressure had been 140 mm. Hg., I found that it had now fallen to 105 mm. Hg. This would seem to indicate that the fall of blood pressure was the result of, and not the cause of, natural sleep (see Chart 1B).

There would appear then to be certain cases of sleeplessness in which a high blood pressure was directly the cause of sleeplessness, and in these cases sleep can be induced by artificially lowering the blood pressure.

There appear also to be cases in which high blood pressure during the waking hours does not necessarily mean sleeplessness, but, as far as my observations go, the blood pressure in these cases always falls after sleep has been established.

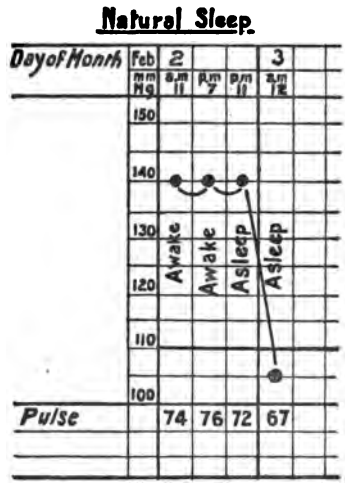


CHART 1B.

Chart 1b.—Shows a convalescent patient with a high blood pressure, whose blood pressure upon first falling to sleep at 11 P.M. was 140 mm. Hg., but an hour later was 105 mm. Hg.

(2) What relation does the morning blood pressure bear to the evening blood pressure?

During the period of convalescence when sleep has returned the sphygmometer shows that the blood pressure as a rule is lower in the evening than in the morning. But it does not follow that because the evening blood pressure does not fall the patient will not sleep. Chart No. 1B shows a convalescent patient whose morning and evening blood pressures registered each 140 mm. Hg., and yet whose blood pressure when asleep at 12 A.M. the following morning was 105 mm. Hg.

When a convalescent patient is relapsing and again tending to become sleepless, the evening blood pressure is generally

higher than that of the morning. Chart No. 8B shows a convalescent patient who slept well from the 28th of January to the 3rd of February, during which period her blood pressure

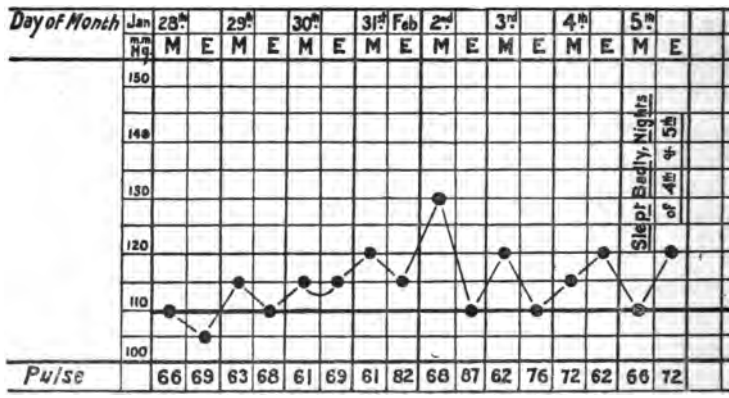


CHART 8B.

was never higher in the evening than it was in the morning. On the 4th and 5th of February the evening pressure was higher than the morning pressure, and on neither night did she sleep well. Chart No. 13A is another example of a relapsing patient with a higher blood pressure.

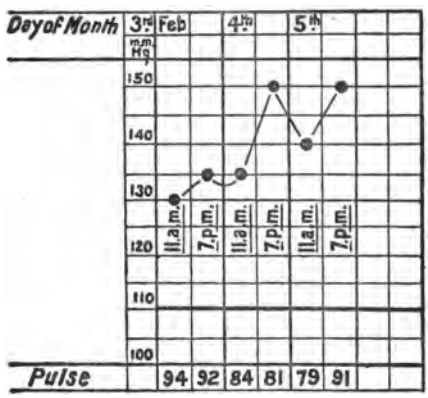


CHART 13A.

To both these rules there are certainly exceptions, probably due to the fact that the blood pressure is a very unstable quantity constantly changing under very slight provocation.

(3) Are there any differences in the blood pressure of the same person asleep under the action of drugs and sleeping naturally?

The only drug experimented with has been paraldehyde.

The sphygmometer readings show that the arterial pressure in any individual person asleep under the influence of paraldehyde sinks lower by about 10 mm. Hg. than it does when that person is sleeping naturally, and the pulsations of the

needle are markedly affected by the respiration in a person sleeping from the effects of paraldehyde, the pressure being increased during inspiration and reduced upon expiration. Chart 6B shows two records of arterial blood pressure during sleep from the same individual. The first record is that of natural sleep when the arterial blood pressure during sleep never fell below 100 mm. Hg.

The second record is paraldehyde sleep, and the pressure fell gradually to 90 mm. Hg.

Chart No. 7B holds a more extended record of arterial pressure in paraldehyde sleep in a patient whose arterial blood pressure was taken during natural sleep upon four different occasions, and upon each occasion was found to equal 100 mm. Hg. Under the influence of paraldehyde, however, the arterial blood pressure fell to 85 mm. Hg. at its lowest, and, as the effects of the drug passed away, the blood pressure gradually rose. At 4.30 P.M. the patient was accidentally wakened when the sphygmometer reading was being taken, and the blood pressure rose to 105 mm. Hg., but shortly afterwards the patient slept again, and then apparently passed into a condition of natural sleep with a pressure of 100 mm. Hg.

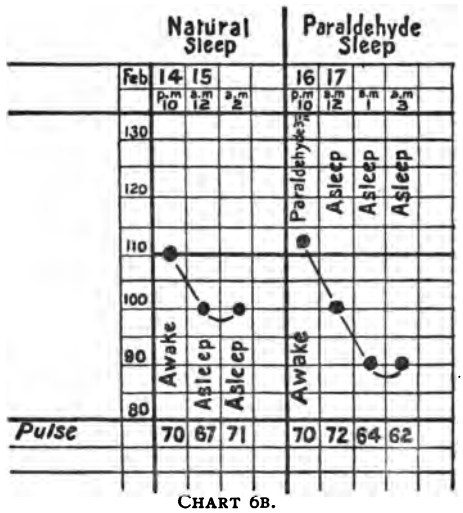


CHART 6B.

Chart 6b.—Record i. shows the fall in blood pressure during natural sleep. Record ii. shows the fall in blood pressure as the result of paraldehyde drug sleep. It will be noticed that the pressure under the influence of paraldehyde has fallen 10 mm. Hg. lower than in natural sleep. These records were both taken from the same patient, who was recovered.

The result of these latter observations is as follows :—

1. Sleep is possible with high blood pressure, but the pressure always falls after sleep is established.

2. That the general blood pressure is as a rule higher in the morning than in the evening in a state of health, and the reverse of this holds good in some sleepless conditions.

3. That in the drug sleep induced by paraldehyde the blood pressure falls at its lowest about 10 mm. Hg. below the

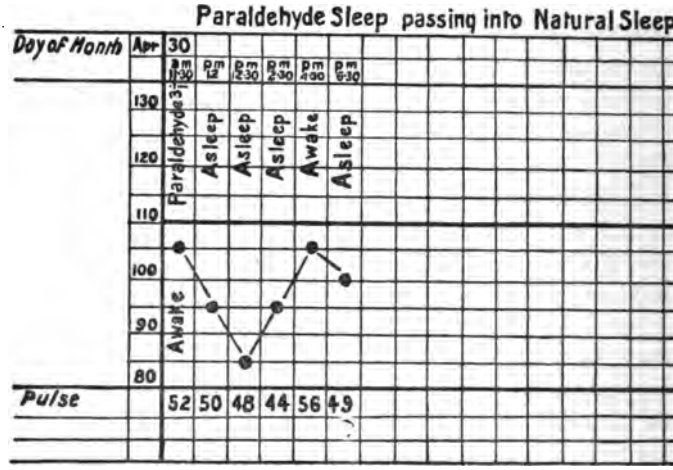


CHART 7B.

Chart 7b.—Shows sleep induced by paraldehyde in a semi-convalescent patient. Paraldehyde ℥i was given at 11.30 A.M. At 12 mid-day the blood pressure had fallen from 105 to 95 mm. Hg.; at 12.30 P.M. the blood pressure was 85 mm. Hg.; at 2.30 P.M. the pressure had again risen to 95 mm. Hg.; at 4.30 the patient awoke and the pressure rose to 105 mm. Hg. Ten minutes later the patient was asleep again, and at 6.30 P.M. the blood pressure was 100 mm. Hg. This was the blood pressure habitual to this patient when sleeping naturally

natural blood pressure of the same person when sleeping naturally, and that as the effect of the drug passes off the blood pressure rises again and eventually passes to the blood pressure habitual to that person in natural sleep; that, in short, drug sleep does pass in some cases into natural sleep.

2. THE ORIGIN AND DEVELOPMENT OF THE UMBILICAL CORD, AND ITS RELATION TO THE AMNION.

By JAMES FOULIS, M.D., F.R.C.P.Ed.

HAS the umbilical cord at any period of its existence a tubular investment of the amnion?

The present investigation was undertaken in order to arrive at a satisfactory answer to this question; for on looking into the subject of the development of the umbilical cord in many of the recently published works relating to the science of midwifery, it was surprising to find how few of the authors of such

works agree on this question—many writers stating that the cord has a tubular sheath of the amnion, and they even describe the steps in the development whereby such an anatomical arrangement is brought about, and also give illustrations in support of their statements, while others, following the good work of Charles Sedgwick Minot, the Professor of Human Embryology in the Harvard Medical School, hold an entirely opposite view. Minot's large work on "Human Embryology" was published in New York in 1892. At page 357 in his book, when describing the development of the umbilical cord, he writes: "The development of the cord shows that it is never covered by the amnion, which, on the contrary, is always separate from the cord proper. This point is important to note because in most text-books the cord is erroneously described as covered by the amnion."

This clear and definite statement by Minot was published in 1892, yet many recent authors have either not noticed it or have passed over it as of no value. Minot's description, however, of the conversion of the ventral string, or Bauchstiel, into the umbilical cord is so difficult to follow that it is not unlikely that many authors have failed to interpret it. Here is Minot's description of the steps in development by which the Bauchstiel is converted into the umbilical cord. At page 357 in his book on "Human Embryology," he writes as follows:

"To convert the Bauchstiel into the umbilical cord, the somatopleure bends down on each side, and finally closing on the ventral side below the allantois, shutting in a portion of the cœlom, and becomes separated from the amnion. The amnion separates from the embryo first, then from the embryonic end of the Bauchstiel, and last of all from the distal end of the Bauchstiel; hence, when the closure of the somatopleure is completed, the amnion arises no longer from the embryo, but only from the end of the cord, where it joins the chorion. The closure of the Bauchstiel forms a long tube running from the embryo to the chorion. The cavity of the tube is part of cœlom, the whole tube is known as the umbilical cord." It must be confessed that it is difficult to follow Minot in this description. But it is quite evident that Minot's important conclusion regarding the relation of the amnion to the cord was the result of his careful observations concerning the development of the human embryo

from its earliest condition; and it is only by a careful study of the development of embryos, both human and animal, that one can get at the truth regarding this interesting question of the relation of the amnion to the umbilical cord.

Unfortunately it is very difficult to obtain human embryos; but such a study in embryology can be well carried out with the aid of embryos of deer, sheep, and pigs, which may be secured in all stages of development. The results obtained from the study of these particular embryos do not differ in any essential point from the results described by His and Minot in their work with human embryos.

My own work has been entirely carried out in connection with the embryos of deer and sheep and pigs, of which I secured a large number.

The first embryo examined by myself was one taken from the uterus of a large red deer. It reached me from the country on the 28th November 1899. As soon as it was removed from the uterus it was placed in a weak solution of bichloride of mercury. As to the age of this embryo it was difficult to speak accurately; but the gamekeeper who sent it to me on my asking him as to its age, wrote as follows: "The chief 'rut' takes place here between the 25th October and 5th November; but always a few does come earlier and a few later. The seasons do not differ much, not more than three or four days between an early and a late season. The does go with young eight months." The embryo measured just six mm. in length, not more, as it lay on its back in a weak solution of bichloride of mercury. It was placed under a specially prepared microscope and then drawn by a skilled artist.

It is represented in Fig. 1 Plate i. just as it appeared under the microscope—and every care was taken to represent all parts accurately as regards size and relation to each other.

The letters A and B point to its head, on which the cerebral vesicles were well marked. The head itself was enclosed in a globular amnion bag, O O, through which the head could be clearly seen. Immediately below the head and touching it, and extending away almost at right angles was the large collapsed yolk sac E, on whose surface numerous blood vessels could be seen.

The yolk sac E was seen to be attached to the primitive intestine by a narrow tube-like structure C, which was scarcely

thicker than the intestinal tube itself. The proximal end of the head within the amnion bag appeared to rest on the yolk sac just at the spot where the yolk sac itself was attached to the primitive intestine, as depicted in the figure.

Above and below the point of attachment of the yolk sac to the intestine the whole body cavity was widely open, and the primitive intestine was seen occupying the middle line as it passed downwards to the caudal end of the embryo, and it preserved the same thickness of tube the whole distance as far down as the origin of the allantois and the allantoic blood-vessels.

The length of the body cavity was about three times its greatest breadth, and at its margins it was boat-shaped.

At the sides of the intestinal tube in its course downwards towards the caudal end of the embryo could be seen two prominent ridge-like structures, F and G. In transverse sections of this embryo afterwards to be described, these ridges were found to be the Wolffian bodies, one on each side of the intestine.

At the extreme caudal end of the embryo the allantois, H H, with its large blood-vessels, K K, could be seen passing outwards over the ventral surface of the embryo on its way towards the chorion, and rapidly expanding transversely into a large bladder-like structure on whose surface could be seen numerous blood-vessels. The blood-vessels K K, and their numerous branches were gorged with embryonic blood-corpuscles.

The allantoic bladder was punctured with a fine needle and a quantity of clear fluid escaped. It is interesting to note in connection with this escape that the primitive intestinal tube and the genito-urinary ducts communicate with the allantoic bladder in such a young embryo. The large blood-vessels, K K, and the allantois itself were enclosed in thick embryonic vascular mesoblastic tissue, which thinned out over the greatly distended allantoic vesicle, as it expanded between the true and false amnion sacs. It was not possible to see any trace of limbs in this young embryo.

The most striking features in the view of this young embryo before us were the comparatively small size of the tubular connection between the yolk sac and the intestinal tube, and at same time, the extreme length of the intestinal

tube itself below that point of communication, and the large size of the open body cavity was very remarkable. The relative sizes and dimensions of all these parts and their proper relation to each other have been carefully preserved in the drawing.

Before proceeding further with our description of these young embryos it is necessary now to say something regarding the cleavage of the mesoblast and the formation of the large body cavity which we have just shortly described, in order that we may become better acquainted with the numerous terms and their significance, used in such an embryological study.

The description of the cleavage of the mesoblast as given in Quain's "Anatomy" at page 36, vol. i. part 1 (Embryology), is so excellent, that we cannot do better than give the very words, and at same time make use of an admirable diagram taken from Minot's work on "Human Embryology" to assist in the description.

At page 36 in Quain's "Anatomy" there is a drawing taken from Kölliker's work representing a transverse section of an embryo chick, in which are represented the medullary folds and the neural canal just closing, also the paraxial mesoblast, and the lateral mesoblast. There is also shown the epiblast, the hypoblast, the primitive aortæ, the notochord and the cœlomic cleavage of the lateral plate of mesoblast.

As regards the cleavage of the mesoblast and the formation of the body cavity, it is thus written: "At a very early period, soon, indeed, after the formation of the neural groove, two important changes begin in the mesoblast. One of these is the cleavage of the lateral mesoblast (which is at first a continuous sheet) into two plates, one of which clings to the epiblast and the other to the hypoblast. The cleft is at first small, but accumulation of fluid within it soon converts it into a cavity, which gradually spreads until the separation is very extensive. The layer of mesoblast which clings to the epiblast, eventually forms part of the body wall, and is known as the *somatopleure*: that which clings to the hypoblast forms eventually part of the wall of the alimentary tract, and is known as the *splanchnopleure*. The cavity between these, which is formed by the enlargement of the original cleft, is the cœlom, or body cavity (pleuroperitoneal cavity of authors)."

Fig. 3 shows the formation of the body cavity, Coe,

by the splitting of the lateral mesoblast into two layers, one of which joins with the epiblast to form the *somatopleure*, Som, while the other joins the hypoblast to form the *splanchnopleure* Spl, which encloses the intestinal tube In, and which

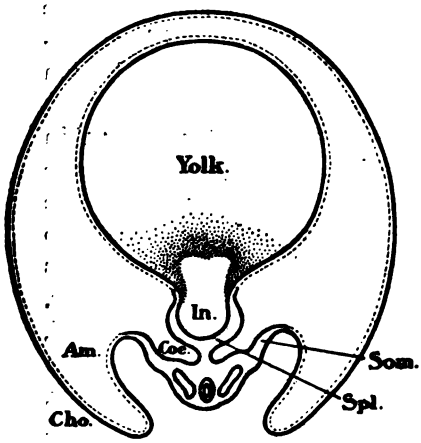


Fig. 3.
Am = Amnion.
Coe = Cœlom.
Cho = Chorion.
In = Intestine.

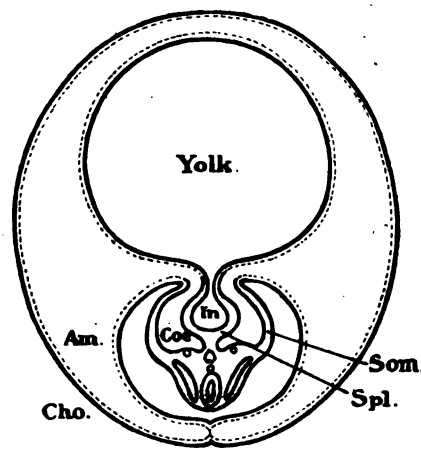


Fig. 4.
Som = Somatopleure.
Spl = Splanchnopleure.
Yolk = Yolk sac.

is still in communication with the yolk sac by the vitelline foramen.

In this figure the lateral somatopleure folds are seen rising up and closing over the dorsum of the embryo; and in Fig. 4 they are represented as having come together in the middle line; and as the line of junction gradually absorbs, the true and false amnion are thus formed. The true amnion, Am, immediately surrounds the embryo. Its cavity is lined by the epiblast. The outer or false amnion, or chorion, is lined by the mesoblast layer, while the epiblast is on the outside. In both figures the dotted lines represent the mesoblastic layers which are always opposed to each other in the encasing membranes. "The mesoblast of the one is separated from that of the other by a space occupied by fluid and continuous with the cœlom, with which, in fact, it remains continuous until the body walls of the embryo have entirely grown round and coalesced on the ventral surface, the final point of coalescence being the umbilicus."

"In the latter stages of gestation the space between the

amniotic and chorionic mesoblast disappears, and these two membranes become loosely united by a jelly-like connective tissue. When the body walls have thus closed in around the yolk sac the latter, greatly diminished in size, lies altogether outside the body of the young animal and is surrounded by the umbilical foramen and communicates with the primitive intestine by means of the vitelline duct, the orifice of communication being termed the vitelline foramen."

Regarding the formation of the allantois, at page 44 in Quain's "Anatomy" we read, "the time of development of the allantois seems to vary very much in mammals; and there is reason to believe that it is found in the human embryo at a very early period. Indeed, the earliest human embryos that have hitherto been described already possess an allantois. In most animals in which its development has been studied, the allantois has been found to begin as a hollow prolongation of the posterior end of the alimentary canal. It soon, however, becomes relatively shifted in position so as to come off from the ventral wall of the hind gut, growing into the posterior extension of the mesoblastic cleft, and eventually into the space between the true and false amnion and carrying along with it its mesoblastic covering.

It is therefore composed eventually of two parts (1) a hypoblastic sac which communicates, at first widely, but afterwards by a narrowed orifice, with the hind gut, and (2) an investment of mesoblast. This last is usually greatly thickened and very vascular, and is directly supplied with blood by two arteries (allantoic or umbilical arteries) which appear at first as a direct continuation of the primitive aortæ. As the allantoic vesicle expands into the cavity of the false amnion it carries the vascular mesoblast along with it, so that this mesoblast is thus brought to the inner surface of the chorion, over which the blood-vessels then spread, so as to convert this hitherto non-vascular membrane into one which is richly supplied with blood-vessels.

The chorion has grown in the form of ramified villi into the substance of the mucous membrane or decidua, even before this advent of the vascular tissue of the allantois, but the chorionic villi now receive blood-vessels, and thus become vascularised, the interchanges between the fœtal and maternal vascular s stems, which are afterwards confined to one region only

of the chorion and decidua—that which forms the placenta—occurring in the first instance over the whole superficies of the ovum.

In the earliest human ova, in which the allantois has been investigated, it is already a tube of hypoblast which forms a direct prolongation of the posterior end of the alimentary canal, and is enclosed in a short stalk of mesoblast by which the posterior end of the embryo is attached to the chorion, and through which, by the allantoic (umbilical) blood-vessels, the chorionic villi are freely supplied with blood.

The stalk in question is not the umbilical cord, since it does not include the stalk of the yolk sac (vitelline duct), which only later becomes bound up with it.

This stalk is termed by His the abdominal stalk (Bauchstiel), the term allantois being by him restricted to the hypoblastic diverticulum."

In our further examination of this embryo, it was then turned over in such a way, that a good view was obtained of its dorsal parts. It was placed under a binocular microscope, and illuminated by means of concentrated light rays, and then drawn by the same skilled artist as before.

The appearance of the dorsal parts of this embryo is well shown in Fig. 2 Plate ii.

The head of the embryo could not be shown, but the globular bag-like amnion, O O, enclosing the head was well seen, and it was found to be continuous with the amnion, which closely invested the whole dorsal surface of the animal. The amnion everywhere as it passed off the dorsal surface was found to be attached to the margins of the large body cavity which was so well seen in the first view of this embryo. The embryo was again turned on to its back, and then the amnion could be traced coming off the dorsal surface everywhere to be attached to the margin of the widely open body cavity, or, as we shall now call it, the widely open umbilical foramen. The large but shrivelled-up yolk sac could be seen passing outwards from under the head towards the left of the animal. The amnion membrane as it covered the dorsal surface was almost transparent, and no blood-vessels could be seen in its substance.

A remarkable linear structure was seen in the substance of the left wall of the embryo. It is indicated by the letter G in

the drawing, and was found to be a large vein gorged with embryonic blood corpuscles coursing downwards from the region of the heart, in the substance of the lateral wall, to be continuous with the large allantoic vein, K, which was connected with the allantoic vascular circulation. On the right side a similar vein was seen. These allantoic or umbilical veins were found to lie external to the allantoic arteries, which are direct prolongations of the primitive aortæ, as they coursed over the allantois at its origin. As they left the lower end of the embryo with the allantois these blood-vessels were found to be encased in thick mesoblastic tissue derived from the lateral walls of the embryo.

It is necessary to direct special attention to the presence of these large veins in the side walls of the embryo. These side walls at this stage of development are known as the lateral somatopleure folds, and it is proper now to describe these large umbilical or allantoic veins as lying in the substance of the lateral somatopleure folds, as they pass downwards from the region of the heart towards the allantoic vesicle, over which they are distributed. In the transverse sections, to be described later on, it will be seen that these large umbilical veins lie in the substance of the somatopleure folds just at the line where the folds turn outwards and become continuous with the amnion as it passes over the dorsal surface of the embryo.

Another very important structure to direct attention to is the rump end of this little embryo, which projects considerably beyond the origin or root of the ventral string or allantoic outgrowth. In the Figure 2 Plate ii., the allantois H H has been drawn well away, so as to expose the rump of the little embryo. The ventral string, in the midst of which is the allantoic diverticulum from the intestine, is often described as a direct extension towards the chorion of the tail end of the embryo. A careful examination of this young embryo before us shows that its rump end projects considerably beyond the point at which the ventral string or Bauchstiel of His leaves the ventral surface of the embryo, and that the amnion membrane as it passes downwards over the dorsal surface embraces the rump end, and then turns forward round the rump to be attached to the very root of the ventral string or Bauchstiel at a point some distance from the distal end of the rump. At this stage there is no appearance of external genital organs, or anus, or tail; but the

whole rump end of such an embryo is tightly encased in the amnion.

A study of transverse sections carried through the root of the ventral string, or Bauchstiel, and through the rump end of the embryo, makes it perfectly clear that it is a mistake to describe the ventral string as a continuation of the tail end of the embryo towards the chorion. The external genital organs, the anus, and tail develop in connection with this rump end of the embryo, but distally to the attachment of the amnion at the root of the Bauchstiel as the latter comes off from the ventral surface of the embryo. In all the early embryos examined, and they were many, this rump end was well marked, and in every case the amnion was found to embrace it closely, and then turn forward some distance to be attached to the root of the ventral string or Bauchstiel. This tail or rump end of the embryo is probably produced at an earlier stage when the *hind gut* is being formed. According to Quain, "all the three layers of the blastoderm are involved in the forward growth and overfolding which produces the head of the embryo, so that a prolongation from the blastoderm cavity, which is, of course, lined by hypoblast, becomes included in the head, and the anterior part of the primitive alimentary canal, or *fore gut*, is thereby produced. Formed in this way, its front end is necessarily blind, and for a long time there is no mouth nor any communication between the fore gut and the exterior of the embryo. The mouth becomes formed later by an invagination from the exterior. Lastly, at the tail end of the embryo a *hind gut* is produced. In most mammals and birds it is produced by a folding over of the tail end of the embryo like that which occurs at the head to enclose the fore gut. The hind gut remains for a considerable time blind, until the anus becomes formed by invagination from the exterior."

This explanation of the formation of the rump end of the embryo, namely, by a folding over of the blastodermic layers at an early stage to enclose a hypoblastic hind gut also explains how it is that the Bauchstiel which contains the tubular hypoblastic diverticulum from the intestine, comes off from the ventral surface of the embryo at some distance from its caudal or rump end, and we know that the external genital organs, the anus and the tail are formed externally to the root of the Bauchstiel; but it also explains how the amnion, after embracing the rump end of the embryo, appears to be

always attached to the very root of the Bauchstiel or ventral string. This rump end of the embryo is formed by the folding over of the caudal end of the embryo. It will be shown as we proceed that the amnion is everywhere an extension of the somatopleure folds, both at the sides, and at the tail and head ends of the embryo. The amnion which embraces the rump end of the embryo is simply an extension of the somatopleure fold in this region as will be afterwards explained.

At this early stage of development the Bauchstiel is entirely mesoblastic in structure; but, of course, has in its substance the hypoblastic tube of the allantois with the allantoic arteries and veins.

At first, as the Bauchstiel or ventral string grows outwards towards the chorion from the ventral surface of the embryo, its mesoblastic tissue and the mesoblastic tissue of the two lateral somatopleure folds are found to be in contact and fused together. The Bauchstiel with its allantoic tube and the vascular mesoblastic tissue of the somatopleure folds then grows outwards to the chorion, while from its root the somatopleure fold gives off the amnion membrane which now closely embraces the rump of the embryo, and which is pushed before it as the rump grows, and then becomes continuous with the amnion encasing the whole dorsal surface of the embryo. The amnion everywhere comes off from the somatopleure folds, both at the sides, and at the head and tail ends of the embryo. Whatever the appearances may be there is no exception to this rule. A study of the transverse sections of this embryo makes this point absolutely clear.

It is now possible to describe the transverse sections referred to. All these sections were carefully photographed, and they have been reproduced by the zinco-collotype process as the best means of giving to us an exact representation of the parts and their relations to each other, as in nature.

Plate iii. Fig. 5 is a transverse section through the young embryo just described, in such a way as to pass vertically down through the large yolk sac. It might be well here to mention that these figures can be seen to greater advantage if looked at through an ordinary hand lens. They come out much better for being thus magnified by a simple lens.

The neural canal, chorda dorsalis, and aorta are well shown. The aorta is almost double in this section. Immedi-

ately below the aorta a short mesentery can be seen attached to the splanchnopleure, which in section is seen to consist of splanchnic mesoblast lined with hypoblast, and the letter I points to the primitive intestine thus formed and still in communication with the yolk sac by a wide vitelline foramen. It is interesting to compare this section with the drawing of the young embryo as seen in Fig. 1 Plate i. The size of the tube of communication between the intestinal tube and the yolk sac is thus made clear. On each side of the mesentery and the intestinal tube, the large Wolffian bodies, W, can be seen, projecting into the cœlom, Coe, or body cavity, which is now widely open.

The genital ducts and glomeruli and tubules in the Wolffian bodies are well seen. The two lateral somatopleure folds, S, one on each side in the section, are beautifully brought out. These somatopleure folds, as we learn by a study of Figs. 3 and 4, are formed at an early stage of development when the lateral mesoblast splits into two layers. The external layer joins the epiblast, and the conjoined membranes now form the somatopleure fold which we see in the sections before us.

Looking carefully at these folds as they leave the body of the embryo to enclose the cœlom, or body cavity, it is easy to see that the somatic mesoblast is in more or less close contact and union with the epiblast. The letter S points to the somatic mesoblast of the somatopleure fold, and the letter E points to the epiblastic layer in union with the somatic mesoblast. In some parts as we follow the somatopleure folds, the epiblast is seen to be partly detached from the somatic mesoblast layer; so that it is perfectly clear that the lateral somatopleure folds now consist of two layers, the somatic mesoblast on the inner side and the epiblast on the outer or external side. Following these lateral somatopleure layers we come to a well-marked bulging out part on their mesoblastic surfaces, just at a point where these folds are about to turn outwards to become continuous with the amnion membrane, O, which can now be traced as passing entirely round the dorsal surface of the embryo. These bulging-out parts are the allantoic or umbilical veins, L, as seen in section. Through the whole series of sections from the head end of the embryo to its caudal extremity, these veins are found to lie in the same position in

the two lateral somatopleure folds. The mesoblastic tissue round these veins is much thickened, as seen in the figures; and just external to the veins, the somatopleure folds can be seen turning outwards and then downwards in such a way as to pass completely round the dorsal surface of the embryo; but it will be noticed that the somatopleure folds, though very thick in the neighbourhood of the umbilical veins, rapidly thin off and become continuous with what appears to be a thin membranous layer, the true amnion, O.

From this description of the manner in which the somatopleure folds are developed, it must be allowed that the amnion, which is a direct continuation of those folds, is morphologically a double layered membrane, consisting of somatic mesoblast and epiblast conjoined. A study of numerous sections of these young embryos makes it perfectly clear that the amnion sac is lined on its inner surface with epiblastic cells, while its outer layer consists of somatic mesoblast. It thus comes about that the true amnion sac is lined with epiblastic cells which are continuous with the epiblastic cells covering all parts of the body of the embryo. In connection with the sections through these umbilical veins it is interesting to observe that the left vein through the whole series of sections appears to be much the larger of the two.

Although the lateral somatopleure folds lie pretty close to the Wolffian bodies in this young embryo, it will be seen that their mesoblastic surfaces are as yet a long distance apart from the mesoblastic surface of the yolk sac with which they ultimately adhere when the cœlom or body cavity is shut off. The section before us shows how widely open is the body cavity, and how large is the umbilical foramen around the yolk sac at this stage of development, while the vitelline foramen is comparatively small.

Fig. 6 represents a section through this embryo in its lumbar region just above the situation where the allantoic outgrowth arises. The intestinal canal is now closed in, and there appears to be no mesentery, but the splanchnopleure, with its hypoblastic lining as the intestinal tube, is well seen in section. The Wolffian bodies are here small but their ducts are visible. The cœlom, or body cavity, is now very deep, but not very wide. The somatopleure folds have approached each other, and their mesoblastic surfaces are nearer

to each other than in the last section. There is no trace of a yolk sac. The umbilical veins, L L, in section are now very well marked, that on the left hand side being particularly so. The somatopleure folds appear to be very thick just as they turn round, external to the veins, to become continuous with the amnion membrane, which, with the aid of a small lens, can be seen to be a double layered membrane.

The next section, Fig. 7, is an important one as showing that the two somatopleure folds have now come in contact with each other, and have fused together around an elongated tubular structure which represents an almost vertical section through the allantoic tube, A, coming up from the cloacal end of the primitive intestine, I. The section has evidently passed vertically down through the allantoic tube as it was coming upwards to pass over the ventral surface of the caudal end of the embryo towards the chorion. This tube is lined with hypoblastic cells continuous with the cells lining the primitive intestine. Note how exceedingly thick is the mesoblastic tissue around the allantoic tube; and with the aid of a small lens it is possible to see the allantoic arteries in section one on either side of the allantoic diverticulum; and external to the arteries, the large umbilical veins are seen in the somatopleure folds in their usual situation. The somatopleure folds, although adhering and fused to the thick mesoblastic tissue around the allantoic tube, can be seen to turn round and outwards and to be continuous with the amnion membrane covering the dorsum of the embryo as before described.

In Fig. 8, which is a vertical section through the Bauchstiel or ventral string, the thick mesoblastic tissue surrounding the allantoic tube is well shown; but the allantoic tube now as it runs horizontally outwards towards the chorion presents in section a tubular outline quite different from that presented in the last section.

The somatopleure folds here seem to be fused to the thick mesoblastic tissue surrounding the cloacal enlargement of the primitive intestine. The cœlom, or body cavity, is here remarkably small. The primitive intestine, I, with its cloacal enlargement and lined with hypoblastic cells, is well marked, and on either side of it there is a section of the large allantoic arteries N, N, which are again seen in the upper part of the figure, just below the section of the allantoic tube. The body of the

embryo has now a solid appearance and is almost nipped off from the Bauchstiel as the latter passes off from its ventral surface; but a study of the figure shows undoubtedly that the somatopleure folds still hold their morphological relation to each other and to other parts of the embryo. For on tracing up the somatopleure folds from their roots just outside the Wolffian bodies it is found that they bend inwards, first of all, and fuse with the mesoblastic tissue around the primitive intestine, in which the large allantoic arteries are seen; then they turn outwards, and in section present a remarkably thick mesoblastic mass, in the midst of which the allantoic arteries are seen just below the allantoic tube; and external to these arteries sections of the umbilical veins are seen in the substance of the true somatopleure folds, which now are just about to turn round and backwards in the usual way, and then thin off to become continuous with the amnion membrane. All this can be clearly made out in these sections; and it would appear as if the splanchnopleure and somatopleure folds at the caudal end of this embryo had never completely separated from each other, as the cœlom between them is reduced to a very small space, and these folds are continuous with each other round the cœlom, just below the situation of the allantoic arteries on either side of the cloacal end of the intestine. It is also very evident from this section that the Bauchstiel, or ventral string, consists for the most part of a thick central mass of vascular mesoblastic tissue surrounding the allantoic diverticulum, and that it is embraced on both sides by the very thick mesoblastic tissue of the two lateral somatopleure folds which then always turn outwards and backwards to give off the amnion membrane.

It is interesting to observe that while in the earlier sections the neural canal is completely closed, in the later ones it is still open, and the epiblast is everywhere seen continuous with its medullary folds.

In Fig. 9, which is a section vertically downwards and transversely through the Bauchstiel, or ventral string and rump end of the embryo, we find that the rump or body of the embryo at this situation is completely separated from the under surface of the Bauchstiel, and that the Bauchstiel is now considerably flattened out transversely. In the upper part of the Bauchstiel the cut tube of the allantois is seen, A. Below it and at each side of it

there is a great mass of vascular mesoblastic tissue. Just below the allantoic tube in the central region of the Bauchstiel are seen the two allantoic arteries, N N, while to the outside of these are seen the large umbilical veins, L L, in the substance of the lateral somatopleure folds just at the point where these folds turn round and backwards and thin off into the amnion membrane, O, which now closely embraces the rump end of the embryo.

Looking carefully at this and the following figure, it would appear as if the two lateral somatopleure folds were for the most part placed below the central mass of the Bauchstiel mesoblast. The apparent change in their morphological relation is probably due to the flattening out of the Bauchstiel; for it must not be forgotten that in the deer the allantoic tube with its thick mesoblastic vascular covering quickly spreads out transversely, between the true and false amnion sacs on its way to reach the chorionic cotyledons.

In this rump end of the embryo, the cœlom or body cavity between the lateral somatopleure folds and the splanchnopleure folds is hardly to be seen, and these folds themselves are united in one undivided mass around the cloacal end of the primitive intestine. In connection with this cloacal enlargement, I, as seen in this figure, it is evident that the hypoblastic lining at the upper part is in the act of forming two tubular structures by a process of invagination. These two tubular structures, completely closed in, are seen in the next figure 10, and, evidently, represent sections of the genito-urinary ducts, which are connected at this stage of development with this cloacal enlargement.

When describing the relation of the rump end of the embryo to the root and under surface of the Bauchstiel, we endeavoured to point out that the amnion membrane was not directly attached to the under surface of the Bauchstiel at its root after embracing the rump of the embryo; but that, embryologically, the mesoblastic surfaces of the two lateral somatopleure folds are fused to the sides and under surface of the mesoblastic Bauchstiel in such a way that the amnion membrane comes off from these folds at the root of the Bauchstiel, and then embraces the rump end of the embryo as a bag-like protrusion, and that morphologically, the amnion is everywhere a continuation of the lateral somatopleure folds. A glance at the figures 9 and 10 will make this statement clear and intelligible; for it is quite evident, from our examina-

tion of numerous sections, that as the lateral somatopleure folds are continued downwards they and the large veins in their substance become attached to and fused with the thick mesoblastic tissue of the Bauchstiel which encloses the allantoic diverticulum with the two allantoic arteries. It would, indeed, appear as if the Bauchstiel or ventral string was an outgrowth of a tubular diverticulum from the primitive intestine with the allantoic arteries in a mass of splanchnic mesoblast, and embraced at its side and under part by the two lateral somatopleure folds in which are the large umbilical veins.

An examination of a whole series of such sections of the caudal end of this embryo proves to us that though in the region of the yolk sac the body cavity or cœlom is very widely open, and that the splanchnopleure and somatopleure folds are widely distant and separate from each other, yet as we proceed in our sections towards the rump end of the embryo, we notice that the body cavity or cœlom becomes less and less wide, and that at last we reach a section in which the margins of the body cavity (see Fig. 7) are in contact and fused around the mesoblast surrounding the primitive intestine. In this case we find that the somatopleure folds are now in contact with the splanchnic mesoblastic tissue surrounding the intestine; and so the cœlom is gradually shut in and reduced in size until we come to sections in which we find the cœlom has almost entirely disappeared.

In Fig. 11, which is a section through the rump end of the embryo at a considerable distance from the root of the Bauchstiel, the amnion membrane, O, or girdle of the somatopleure fold, is quite a thick structure as it passes round and embraces the rump. The epiblast is well-marked, and is seen in continuity with the medullary folds of the neural canal. In this section no trace of an intestine can be seen; and only mesoblastic plates in an undivided condition are seen within the epiblastic ring. At this region the allantoic tube has flattened out transversely in the flattened ventral string, and there is no trace of the lateral somatopleure folds passing off into an amnion membrane. The ring of amnion around this section of the rump is without doubt a part of the amnion arising in connection with the caudal fold of the somatopleure at the root of the Bauchstiel in Figs. 9 and 10.

Though the somatopleure folds appear now to lie below the thick mesoblastic Bauchstiel owing to the great expanse

of the latter transversely, yet it can be seen in the sections, that these lateral somatopleure folds give off the amnion membrane in this region just as in other situations. It is in this region that the amnion is given off from the somatopleure fold at the root of the Bauchstiel, and it encases the whole rump and is pushed before it as the rump grows and becomes larger.

In Fig. 2 Plate ii. the rump end of the embryo from which these last sections have been prepared is well shown; and it will be at once seen that the ventral string or Bauchstiel is not a continuation of the tail end of the embryo, but that it is a mesoblastic out-growth from the ventral surface of the embryo with the allantoic arteries and tube within its central part. This mesoblastic out-growth is embraced by the two lateral somatopleure folds with the large allantoic veins in their substance. These somatopleure folds gradually come to lie at the side and under surface of the Bauchstiel as the ventral string or Bauchstiel expands laterally, and as it passes outwards over the ventral surface of the rump end of the embryo. The amnion membrane everywhere is an extension of these folds in all situations.

The last section, Fig. 12, which I shall describe is one of the last of the series. The section was made by the knife passing vertically downwards through the widely expanded Bauchstiel and through the very rump end of the embryo lying below it. A thick ring of somatopleure fold, O, is here seen around a shaving cut off the enclosed rump. This ring of somatopleure is part of the bag which surrounds the whole rump end of the embryo at this stage, and it is derived from the caudal somatopleure fold at the root and under surface of the Bauchstiel. As the general amnion sac later on becomes distended with fluid this bag of somatopleure fold round the rump is also distended, and the somatopleure fold is stretched out into the thin amnion membrane continuous with that which everywhere encloses the body of the embryo.

It is interesting to note here the distance that exists between the under surface of the Bauchstiel and the mesoblastic ring surrounding the rump shaving. The Bauchstiel, or ventral string, as it lies above this ring is seen now to be very widely expanded and flattened out, and in the midst of it is seen (in section) the widely dilated allantoic tube

lined with hypoblastic cells. The mesoblastic tissue around this allantoic bladder is very vascular, and in it are seen numerous arteries and veins.

Let us now consider a little more carefully the origin and development of

The Bauchstiel, Abdominal Stalk or Ventral String, and its relation to the Somatopleure Folds and Amnion

One hundred and thirty-five photographs of sections through this young embryo were placed in their proper relation to each other, and fixed on a large piece of cardboard for study. The sections examined passed, first of all, through the yolk sac and its attachment to the primitive intestine, then through the middle part of the body, then through the lumbar region, and lastly through the Bauchstiel and rump end of the embryo.

The following conclusions were drawn from such a study.

At an earlier stage of development than this embryo presents, there must have been a folding over of the three primary layers of the embryo to produce the head fold by which the fore gut was included in the head region as a blind tube. The cephalic somatopleure fold can be traced in this region bending upwards and backwards over the head of the embryo to be continuous with the general amnion sac. This cephalic fold of somatopleure starts from the cephalic end of the umbilical foramen. The two lateral somatopleure folds can be traced bending back and then turning round the dorsal surface of the embryo as the amnion membrane. They border the lateral parts of the umbilical foramen. At the caudal end of the embryo there must also have been a folding over of the primary layers to produce the tail fold which encloses the blind hind gut; and in the sections it is easy to follow the bending back of the caudal somatopleure fold which, as a thick layer, turns backwards and downwards to embrace the rump end of the embryo produced by the tail fold. All these somatopleure folds, the cephalic, lateral, and caudal folds, are directly continuous with each other round the margin of the large umbilical foramen; and while the cephalic fold turns upwards and backwards to embrace the head, the somatopleure folds in all other parts turn downwards and backwards to embrace the dorsal surface and rump end of the embryo in one continuous sheet.

In the cleft, at the caudal end of the umbilical foramen, at the region where the caudal fold of somatopleure turns backwards to embrace the rump end, a tubular outgrowth from the primitive intestine makes its appearance. This tubular outgrowth, accompanied by the two prolongations of the primitive aortæ, and encased in a mass of mesoblastic tissue, grows outwards in this cleft between the margins of the umbilical foramen, and then turns backwards in such a way that the under surface of this mesoblastic outgrowth lies in contact with the upper mesoblastic surface of the caudal somatopleure fold to which it is attached. This mesoblastic vascular outgrowth is known as the Bauchstiel, or abdominal stalk of His. It is also called the ventral string. It has no epiblast covering at any part, and consists entirely of mesoblastic tissue.

The large allantoic or umbilical veins, in the sections, in the substance of the lateral somatopleure folds, are always seen just internal to that outward bend of the somatopleure folds which takes place when the folds turn round and backwards to pass round the dorsum of the embryo as the amnion membrane. The sections near the caudal cleft of the umbilical foramen show the folds are greatly thickened in this region, and that the large umbilical veins lie in a very thick mass of mesoblastic tissue. Following the sections onwards, it is seen that the thick mesoblastic tissue of these lateral somatopleure folds gradually approach and grasp and adhere to the thick splanchnic mesoblastic tissue surrounding the hypoblastic tubular outgrowth from the intestine; and the sections further show that the umbilical veins now pass inwards, and, lying external to the allantoic or umbilical arteries, become involved in the Bauchstiel or ventral string, as it grows outwards over the caudal somatopleure fold towards the chorion. While the allantoic or umbilical veins thus go inwards to form part of the Bauchstiel which is now like a tail prolongation of the embryo, but lying superficial to its rump end, the somatopleure folds which are very thick in this region, bend outwards and downwards, as in other parts, and being directly continuous with the caudal somatopleure fold they appear to sweep under the Bauchstiel, but it is clear that this appearance is caused by the Bauchstiel broadening out and then passing over the caudal fold of the somatopleure to which it is fused at this point. The caudal fold then separates from the Bauchstiel and passes at once

round the rump end, and is everywhere continuous with the amnion round the dorsal surface of the embryo.

In leading works on Embryology such as those of His and Minot, we read that the amnion is *attached* to the sides and to the root of the Bauchstiel. Whatever the appearances later on may be between the Bauchstiel and amnion membrane, they first come in contact and fuse to each other in the way I have described; namely, by the under mesoblastic surface of the Bauchstiel attaching itself to the upper mesoblastic surface of the caudal fold of somatopleure, and this attachment takes place before the caudal fold is stretched out into the thin amnion membrane which it subsequently is. The caudal fold is at first quite a thick layer of somatopleure as it lies under the Bauchstiel and embraces the rump: all the folds are remarkably thick as they first come off from the margins of the umbilical foramen, and there is little space between them and the body of the embryo when first formed. This sac, which embraces the whole embryo, and known later on as the sac of the amnion, is attached only to the sides of the umbilical foramen and to the under surface of the Bauchstiel, in the way I have described. The attachment of the under surface of the Bauchstiel to the mesoblast of the caudal fold is not at first an extensive one; for the fold quickly turns round and embraces the rump, while the vascular Bauchstiel with its allantoic tube passes straight out to reach the chorion; but it is a most important one, as will be demonstrated when we are considering the conversion of the Bauchstiel into the true umbilical cord.

The outer surface of the amnion sac is entirely mesoblastic; while the lining of the sac everywhere is epiblastic and is continuous with the epiblastic covering of the enclosed embryo.

The space between the two epiblastic surfaces is at first very slight, but as the fluid between them increases, not only does the space enlarge, but the margins of the umbilical foramen to which the amnion is attached are pulled upon and raised up in the form of a tubular prolongation of the body of the embryo; and at this stage of development the umbilical foramen has considerably diminished in size around the yolk sac. While the outer surface of this tubular prolongation of the body wall is epiblastic and lies within the amnion sac, the inner surface which forms the wall of the cœlomic tube and encloses the yolk sac, or vitelline duct, is mesoblastic.

It is very important to notice that the surface of the Bauchstiel which lies in the caudal part of the umbilical foramen, and which faces the mesoblastic surface of the yolk sac at the cephalic end of the umbilical foramen, is entirely uncovered by amnion, and consists of splanchnic mesoblast. The amnion, as we have so often stated, is always continuous with the somatopleure folds at every part; and as the folds come off from the sides of the Bauchstiel, after the amnion sac is distended with liquid, by which means the Bauchstiel is drawn out, both at its sides, and lengthwise; so it is found by a careful study of the sections, that the amnion comes off from the sides of the Bauchstiel at a line external to the line of the umbilical veins as they course along the Bauchstiel towards the chorion; but the Bauchstiel in its middle part, within the line of the umbilical veins, is entirely uncovered by amnion.

Having thus described the young deer embryo, Plates i. and ii., and all its parts in their natural relation to each other, as seen in the front and dorsal aspects, and having directed special attention to the large widely open umbilical foramen, and to the comparatively small tubular connection between the yolk sac and the upper part of the primitive intestine; and, also, having described the rump end of the little embryo at this stage of development, we then shortly described several sections, in series, through its body, in order that we might bring out more clearly the origin, structure, and relations of the somatopleure folds to the amnion sac enclosing the body, and to the Bauchstiel, ventral string, or abdominal stalk, which connects the embryo to the maternal uterine surface.

We shall now, lastly, describe the steps in the further development by which the Bauchstiel or ventral string, is converted into the true umbilical cord.

It will be necessary, however, in the first instance, to examine very shortly the appearances presented by two other deer embryos, as seen in Plates vii. and viii., which are somewhat older than the one already described.

The embryo first described, as seen in Plates i. and ii., as it lay on its back, was not in any way doubled up, so as to approximate its head and tail ends, although there was a marked head and tail fold enclosing the fore and hind gut:

but in the embryos as seen in Plates vii. and viii., there was a well marked approximation of the head and tail ends towards each other, as is well seen in Plate viii. where the embryo is lying on its side and the dorsal line is curved.

One of these embryos was placed on its back in such a way as to expose its ventral surface, which was then examined under a binocular microscope; and it was then possible to look down into the umbilical foramen. On comparing the umbilical foramen in this embryo, Fig. 13, with the umbilical foramen in the first embryo, Fig. 1, we see that in the first the foramen is very large and widely open, while in the second one it is closing in around the vitelline duct of the yolk sac, C. The whole animal is enclosed in an amnion bag which comes off from the umbilical foramen. In the drawing before us, Plate vii., it is difficult to see the amnion over the head region; but it can be seen somewhat distended with fluid between the letters M and D on the left side, and at the letter H on the right side of the animal, bulging out, as it were, and then passing inwards to the margin of the umbilical foramen on the one side, and then passing outwards over the whole body and rump end. As the amnion passes over the rump end, it turns forward, and appears to be attached to the sides and to the under surface of the Bauchstiel at its root. The letter M points to the first trace of a hind limb, and it is enclosed within the amnion sac. The large allantoic veins and arteries, as they course along the ventral surface of the Bauchstiel, H, are indicated by the letter K. Between the line of these blood-vessels, in the centre, can be seen the Bauchstiel mesoblastic surface which is uncovered by amnion.

The next embryo, Fig. 14, as seen in Plate viii., was a very beautiful object. The dorsal curve was very marked, as it lay on its side, and the head and tail ends were drawn towards each other. A good view was obtained of its head, A, and the branchial clefts were well marked, the heart, B, was quite distinct, and the large liver, D, could be traced bulging out the body of the embryo. The whole dorsal surface was encased in a tight-fitting amnion sac, which could be traced passing downwards over the head to be attached to the cephalic margin of the umbilical foramen. As it passed downwards this head fold of the amnion, N, was in contact with the shrivelled up yolk sac, C, whose vitelline duct could be

seen passing into the body of the embryo through the cœlomic opening of the umbilical foramen. The Bauchstiel, L, was a well-marked string-like structure. It was cut transversely across close to the rump end of the embryo; and then it was possible to see in the centre of the cut surface, as indicated by the letter M, the well-marked tubular orifice of the allantoic vesicle. The letter L points to the situation of the allantoic tube itself as it lies enveloped in a thick mesoblastic covering, between the line of the large blood vessels, veins and arteries, which go out towards the chorion, as indicated by the letters K K. The amnion membrane somewhat distended by fluid could be seen at the sides of the embryo, H, and then, after embracing the rump end, it turned sharply upwards to be attached to the under surface of the Bauchstiel at its root.

Between the letters H and K, this part of the amnion passing upwards to be attached to the under surface of the Bauchstiel at its root is well seen.

In Plate ix. we have very carefully represented the appearances seen in the case of a young sheep embryo, as it lies completely encased in the amnion sac which is everywhere distended with fluid.

The letter A points to the head. Below the head the heart and liver are seen. The letter B points to the true umbilical cord and to the large umbilical vein in its substance. The letter C points to the shrivelled up yolk sac which appears to pierce the umbilical cord at its front part. The yolk sac now lies quite outside the sac of the amnion. The letter N points to that part of the ventral string which is now entirely outside the amnion sac after the umbilical foramen has completely closed in around the vitelline duct of the yolk sac. The letter M points to the hind limb and tail within the amnion; and the letter O points to the general amnion sac filled with fluid to distension, and now attached to the embryo only at the distal end of the true umbilical cord at a point where the vitelline duct appears to pierce and enter the front part of the umbilical cord.

Looking at the figures in Plates i., ii., vii., viii., we may now ask, how is the Bauchstiel, or ventral string, whose origin, development and relations we have described, converted into the young umbilical cord, B, which is so well seen in the embryo before us, in Plate ix?

The Conversion of the Bauchstiel into the True Umbilical Cord

Bearing in mind all we have described regarding the origin, development and relations of the somatopleure folds to the umbilical foramen, and to the vascular Bauchstiel, or ventral string, it will be now easy to follow the steps in development which take place when the umbilical foramen closes in around the yolk sac and the mesoblastic Bauchstiel, and when the whole amnion sac is widely distended into a globular bag whose only attachment to the body of the embryo is at the margins of the umbilical foramen.

As the first result of the distension by fluid of the amnion sac, which is continuous everywhere with the somatopleure folds around the umbilical foramen, the amnion membrane is raised up from off the entire surface of the body of the embryo, and its attachment to the whole umbilical foramen is drawn out and put upon the stretch ; at the same time, explain it how we may, the head and tail ends of the embryo are approaching each other over the umbilical foramen, which is now gradually closing in around the yolk sac and the Bauchstiel, which are within its cœlomic tube, Plate x. The opposing cœlomic surfaces of the umbilical foramen are mesoblastic, and they adhere closely and tightly to each other where they come in contact, and to all parts within the tube ; and as the fluid distension proceeds, the amnion drags upon its attachment to the umbilical foramen in such a way that a distinct tubular process is drawn out from the body of the embryo below the amnion, and it embraces the vitelline duct and the Bauchstiel, which last contains in its mesoblastic substance the remains of the allantoic tube, the allantoic arteries and veins.

This Bauchstiel portion of the tubular process thus drawn out, forms a large part of the young umbilical cord thus produced. The umbilical cord thus formed is within the amnion sac, and the amnion sac is continuous with, and attached to the distal end of the cord. The cœlomic surface of the now closed-in umbilical foramen turns outwards as it is continuous with the outer mesoblastic surface of the amnion, while the epiblastic surface of the young umbilical cord is reflected over the inner surface of the amnion sac.

However long the true umbilical cord may ultimately become, it always lies within the amnion sac, and at its distal

end the outer mesoblastic surface of the amnion sac turns inwards to be continuous with the cœlomic surface of the original umbilical foramen, which now tightly grasps the much drawn-out and shrivelled-up yolk sac or vitelline duct.

However distant the amnion sac may be in the later stages of development from the body wall of the embryo, it arose at first in direct continuity with the somatopleure folds, which formed the margins of the umbilical foramen in the early embryo. The somatopleure folds at first, as they were continuous with the margins of the umbilical foramen, were very thick, and the amnion bag only becomes a large thin-walled membranous sac as the result of extreme stretching of the folds by fluid distension.

In connection with this origin of the amnion from the thick somatopleure folds, it is very interesting to observe that when we cut away the entire amnion bag from its attachment to the umbilical cord at its distal end, we find the amnion is very thick at the line of section, though the amnion bag elsewhere may be distended into a thin membranous sac.

Whatever prolongation there is of the vascular ventral string or Bauchstiel outside of the amnion sac, it is not the true umbilical cord which is always within the amnion sac.

The allantoic arteries and veins, along with the remains of the allantoic tube, as they lie in a mass of embryonic mesoblastic tissue are found in the posterior part of the young umbilical cord, and the mesoblastic tissue enclosing these structures is fused to and inseparable from the fold of somatopleure, which we described as the caudal somatopleure, and which is now continuous with the general somatopleure fold in the wall of the young umbilical cord.

However much the embryonic caudal fold of somatopleure, which was at first attached to the Bauchstiel at its root, and which is now recognisable as the amnion membrane coming off from the posterior part of the young umbilical cord, may appear to be drawn away from its original line of attachment (as in Plates ix. and xi.), such a change in position is only what was to be expected as the result of the distension of the amnion sac by fluid, and the consequent dragging outwards of all parts to which the early amnion bag was attached. Thus, if we look at the young umbilical cord, B, Fig. 15, Plate ix., we see the amnion membrane now comes off from it at a considerable distance from the body of the embryo, though, at first, the

amnion was attached to the embryo along the margin of the widely open umbilical foramen, where it was continuous with the early somatopleure folds, which now, of course, form part of the wall of the umbilical cord.

In a horizontal section through the young umbilical cord and amnion at its distal end, at a line where the amnion turns round from its attachment to the cord, the following appearances are presented. In outline the young cord is more or less oval, Fig. 18. On its outside is the epiblastic layer E, which is reflected over the entire inner surface of the amnion bag. At its front part the remains of the vascular yolk sac, K, with its hypoblastic lining, H, is well seen, and this is surrounded on all sides by the mesoblastic wall, D, or cœlomic surface of the original umbilical foramen, M. F points to the cephalic somatopleure. Posterior to the remains of the shrivelled-up yolk sac is a mass of thick embryonic mesoblastic tissue, B, in which are the remains of the allantoic tube, A, with its hypoblastic lining. Below and to the sides of this tube are the allantoic arteries, N, while external and slightly above them are the umbilical veins, L.

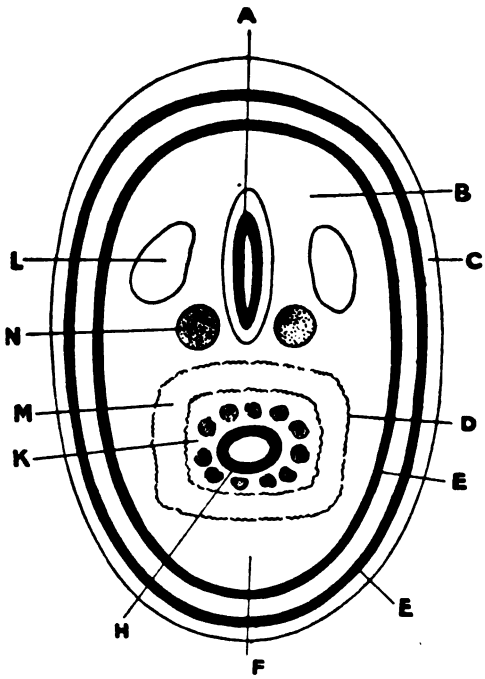


Fig. 18.
Horizontal section through the young umbilical cord and amnion at its distal end.

The vascular structures are enclosed in a thick mass of embryonic mesoblastic tissue which makes up the chief part of the young umbilical cord. It is not possible to detect any line of demarcation between this mass of Bauchstiel tissue and the mesoblastic surface of the embryonic caudal somatopleure fold which now forms the outer wall of the cord. In section, the amnion mem-

DESCRIPTION OF PLATES AND FIGURES.

PLATE I.

FIG. 1.—An embryo deer, 6 mm. in length, lying on its back.

A B Head
C Yolk sac, joining the intestine.
D Intestinal tube at point of junction.
E Large collapsed yolk sac.
G F Wolffian bodies.
H H Large allantoic bladder, covered with vascular mesoblast.
K K Allantoic blood-vessels.
O Amnion.

Note the large widely open umbilical foramen and the small tubular communication between the yolk sac and the primitive intestine at this stage of development, and the length of the intestinal tube.

PLATE II.

FIG. 2.—The same embryo. Its dorsal surface is now seen.

C Collapsed yolk sac.
G Large vein gorged with embryonic blood corpuscles, in the left lateral somatopleure.
H Allantois with blood-vessels, drawn well away from the rump end of the embryo.
K K Large allantoic blood-vessels.
O Amnion.
S Somatopleure tissue covering the allantoic blood-vessels.

Note the rump end of the embryo projecting considerably beyond the point at which the ventral string, or allantoic outgrowth, leaves the ventral surface of the body. The rump end is tightly encased in the amnion.

PLATE III.

FIG. 5.—Microphotograph of section through this embryo at the point where the yolk sac joins the intestine (see FIG. 1).

C O E The cœlom, or body cavity.
E Epiblast detached in some places from somatic mesoblast.
I Primitive intestine lined with hypoblastic cells and in communication with the yolk sac.
L Large umbilical vein in lateral somatopleure, on both sides.
O Amnion—the direct continuation of the somatopleure folds.
S Somatopleure folds continuous with amnion passing round embryo.
W Wolffian body.
Y Yolk sac.

FIG. 6.—Microphotograph of section which passed transversely through the lumbar region of this embryo.

C O E The cœlom, or body cavity.
E Epiblast.
I Primitive intestine lined with hypoblastic cells. There is little or no mesentery here.
L Large vein in the lateral somatopleure. The left vein is very large.
O Amnion.
S Somatopleure fold. Just external to the vein on each side the fold is very thick.
W Wolffian body.

Note—the somatopleure folds in this section are close to each other.

PLATE IV.

FIG. 7.—Microphotograph of section passing vertically downwards through the allantoic outgrowth, and through the body of the embryo at the point where the tube of the allantois comes upwards out of the body.

A Allantoic tube lined with hypoblastic cells.
C O E The cœlom, or body cavity, here closed in.
E Epiblast.
I Primitive intestine lined with hypoblastic cells.
L Allantoic, or umbilical veins, in the wall of the lateral somatopleure folds.
N Allantoic artery—one on each side of the allantoic tube.
S Somatopleure folds. Their thick somatic mesoblastic surfaces in this region have now fused with the thick vascular mesoblast (splanchnic) enclosing the allantoic tube, thus shutting in the cœlom in this region.
O Amnion.
W Wolffian body. Small in this region.

FIG. 8.—Microphotograph of section passing vertically downwards through the Bauchstie , or ventral string, and through the body of the embryo.

A Allantoic tube, lined with hypoblastic cells, passing out horizontally towards the chorion.
C O E Cœlom, here very small.
E Epiblast.
I Intestinal tube, lined with hypoblastic cells.
N N Allantoic arteries, just below allantoi c tube.
O Amnion.
S Somatopleure folds.
W Wolffian body.

Note.—The Bauchstiel in this section consists of the allantoic tube, with allantoic arteries, in the midst of a mass of splanchnic mesoblastic tissue embraced on both sides by the thick lateral somatopleure folds in which are the allantoic veins.

PLATE V.

FIG. 9.—Microphotograph of section through the Bauchstiel and rump end of the embryo, just at the point where the caudal somatopleure fold, under the Bauchstiel, turns round to embrace the rump end of the embryo. The epiblastic surface of the rump, as seen in the section, is here separated from the epiblast lining the inner surface of the caudal somatopleure fold.

A Allantoic tube in section.
B Part of allantoic bladder.
E Epiblastic lining of amnion sac. The letter also points to epiblast on the rump.
I Cloacal enlargement of intestinal tube in which an invagination of the hypoblast is taking place to form two tubes—genital ducts.
L Allantoic vein.
N N Allantoic arteries just below allantoic tube.
S S Somatopleure folds.
O Amnion.
W Wolffian body.

FIG. 10.—Microphotograph of section through Bauchstiel and body of the embryo at rump end—very much the same as in section, Fig. 9, but in Fig. 10 the invagination of the hypoblast in cloacal enlargement of intestinal tube is completed, and two tubes in section are seen.

A Allantoic tube lined with hypoblast cells.
B Part of allantoic bladder.
E Epiblast lining amnion sac and covering body of embryo.
I Intestine.
L Large allantoic vein.
N N Allantoic arteries, just below allantoic tube.
O Amnion.
S S Somatopleure folds forming amnion sac and lined with epiblast.
W Wolffian body.

PLATE VI.

FIG. 11.—Microphotograph of section through flattened out or expanded Bauchstiel, and rump end of embryo, which is surrounded with a thick covering of somatopleure fold, or amnion membrane.

A Expanded allantoic tube.
B Part of allantoic bladder.
E Epiblast on surface of rump section. The epiblast also lines the ring of amnion round this rump section.
L Allantoic vein.
N N Allantoic arteries.
O Amnion.
S Ring of somatopleure, or amnion, round the section of the rump. It is very thick.

FIG. 12.—Microphotograph of section through Bauchstiel and rump, at the extreme end of the embryo—only a shaving of the rump end is seen within the amnion covering which is here very thick.

A Allantoic tube widely expanded.
B Part of allantoic bladder.
L Allantoic vein.
N Allantoic artery.
S Ring of somatopleure round the end of the rump of embryo.

PLATE VII.

FIG. 13.—A deer embryo lying on its back, and so placed as to show the Bauchstiel or ventral string and umbilical foramen now rapidly closing in round the vitelline duct of the yolk sac.

A Head, covered by amnion membrane.
C Yolk sac, the vitelline duct is seen passing into the cœlom or body cavity through the closing in umbilical foramen.
D The vitelline duct entering the umbilical foramen.
H H The Bauchstiel or ventral string.
K K Umbillical blood-vessels, in the ventral string.
M First trace of a hind limb, enclosed by the amnion.

PLATE VIII.

FIG. 14.—A deer embryo placed on its side so as to show the marked dorsal curve which takes place when the head and tail ends approximate over the umbilical foramen.

A Head.
B Heart.
C Yolk sac whose vitelline duct can be seen entering the umbilical foramen.
D Liver.
H Amnion on the side of the embryo.
K K Umbilical blood-vessels of the Bauchstiel. Between the line of these blood-vessels the Bauchstiel in the centre is uncovered by amnion.
L The Bauchstiel.
M Cut allantoic tube in centre of Bauchstiel.
N Amnion membrane covering the head, and attached to embryo at the cephalic margin of the umbilical foramen.

PLATE IX.

FIG. 15.—The embryo of a sheep. The amnion sac round the embryo is distended with fluid, and it is attached only to the distal end of the true umbilical cord, while the ventral string, outside the amnion sac, with its umbilical blood-vessels passes onwards to the mesoblastic surface of the chorion.

The young umbilical cord is seen within the amnion sac, and the vitelline duct appears to pierce the amnion on the anterior surface of the young umbilical cord.

A Head of embryo.
B The young umbilical cord. The letter *B* points to a large umbilical vein in the cord, seen through the amnion.
C Yolk sac. The vitelline duct is embraced by the closed in umbilical foramen at the front part of the umbilical cord at its distal end. The yolk sac now lies entirely outside the amnion sac.
M Hind limb.
N The ventral string outside amnion sac, consisting of mesoblastic tissue with the umbilical blood-vessels and remains of allantoic tube, passing out towards the chorionic villi.
O Amnion sac tightly distended with fluid, and attached only to the distal end of the true umbilical cord.

PLATE X.

FIG. 16.—This drawing, as the result of many observations, was designed to show the manner in which the umbilical foramen closes in around the yolk sac and the root of the Bauchstiel, both of which structures are within the cœlomic tube of the umbilical foramen. The Bauchstiel in the centre, within the line of the umbilical blood-vessels, is mesoblastic, and is uncovered by amnion. The amnion is in connection with the somatopleure folds attached to the embryo only at the margin of the umbilical foramen, and to the side and under surface of the Bauchstiel at its root, as already described.

As the cœlomic tube gradually closes in on the yolk sac and the Bauchstiel, the amnion membrane is at same time raised off the surface of the embryo by fluid distension, and there results a tubular process of somatopleure, below the raised up amnion, around the margin of the umbilical foramen, whose outer surface is epiblastic. This tubular process whose outer surface is epiblastic, as it lies below the distended amnion, is the first trace of the true umbilical cord; and at its distal end the amnion is attached to and continuous with the somatopleure folds which constitute its walls.

A Allantoic tube in centre of vascular Bauchstiel.
B Bauchstiel. The central part between the line of blood-vessels is uncovered by amnion.
C Amnion sac encasing the whole embryo and attached to the embryo only at the margin of the umbilical foramen, and to the sides and under surface of the Bauchstiel at its root.
D Cœlomic mesoblastic surface of the umbilical foramen continuous with the outer mesoblastic surface of the amnion.
E Epiblast covering entire surface of the embryo and then reflected, at the umbilical foramen, over the entire inner surface of the amnion sac.
F F Outer mesoblastic surface of amnion.
K Yolk sac with vitelline duct.
N Umbilical blood-vessels.

PLATE XI.

FIG. 17.—Drawing of a sheep's embryo so prepared as to show the relation between the young umbilical cord and the amnion, the Bauchstiel and yolk sac. The entire amnion membrane has been cut away except a long narrow strip *O O*, which is attached to the distal end of the umbilical cord.

A Head of embryo.
B Bauchstiel outside the amnion sac, with the umbilical blood-vessels, on its way to the chorion. Attached to the under surface and to the sides of the Bauchstiel at this part is the caudal fold of the somatopleure, known now as the amnion, whose under surface is covered with the epiblastic layer reflected from off the umbilical cord which is within the sac.
E Yolk sac whose vitelline duct is tightly embraced by the cœlomic mesoblastic surface of the closed in umbilical foramen. The vitelline duct in the umbilical tube lies against that surface of the Bauchstiel, in the centre, which is uncovered by amnion membrane.
F points to the thick edge of the cut amnion membrane, which is always thick near to its attachment to the distal end of the cord, because the amnion here is a continuation of the thick somatopleure folds, which form the wall of the umbilical foramen.
G The tail of the embryo, in this case lying in a vertical groove on the posterior wall of the umbilical cord.
L The umbilical cord, whose outer epiblastic surface is reflected over the entire inner surface of the amnion. The letter points to vein.
O O Amnion membrane attached to the umbilical cord at its distal end.

PLATE XII.

FIG. 19.—Microphotograph of vertical mesial section through a deer embryo to show the vitelline duct and intestinal tube in the centre of the young umbilical cord, which is within the amnion sac.

B Bauchstiel outside amnion sac with its blood-vessels passing out to the chorion.
I Intestinal tube in connection with vitelline duct, in the umbilical cord.
M Cephalic fold of amnion in direct continuity with the cephalic fold of the somatopleure.
O Amnion passing round the embryo.
S Caudal fold of somatopleure at under-surface of Bauchstiel continuous with the amnion.
Y Vitelline duct.

FIG. 20.—Microphotograph of vertical mesial section through a deer embryo.

A Allantoic tube remains.
B Bauchstiel.
M Cephalic fold of amnion continuous with the cephalic fold of somatopleure.
O Amnion sac round embryo.
S Caudal fold of somatopleure, below Bauchstiel, continuous with the amnion.
Y Yolk sac, vitelline duct.
W Vertical section of Wolffian body.

Note the line of adhesion between the somatic mesoblast of the cephalic somatopleure fold and the anterior surface of Bauchstiel in this section of the young umbilical cord. A fold of the intestine points to it.

PLATE XIII.

FIG. 21.—Microphotograph of section which passed horizontally through the body and umbilical cord of sheep's embryo.

A Remains of allantoic tube.
B Bauchstiel, outside amnion sac.
C O E Cœlom.
I Folds of intestine in the cœlom.
N Allantoic artery.
O Amnion membrane passing round body of embryo and continuous with the thick lateral somatopleure folds from the distal end of the cord.
S Lateral somatopleure folds turning round and backwards after closure of the umbilical foramen, to become continuous with amnion membrane.
Y Yolk sac outside amnion sac.

FIG. 22.—Deer embryo enclosed within the tightly distended amnion sac, almost natural size. The amnion is tightly distended with fluid, and is attached only to the distal end of the umblical cord, while the vascular ventral string or Bauchstiel, outside the amnion sac, spreads out over the inner surface of the chorion to distribute its blood vessels to the chorionic cotyledons.

B Bauchstiel or vascular ventral string.
H Head of embryo within the amnion sac.
O O Tightly distended amnion sac.
T Tail of embryo.
Y Vitelline duct outside the amnion sac at front part of umbilical cord.

PLATE I.

FIG. 1.

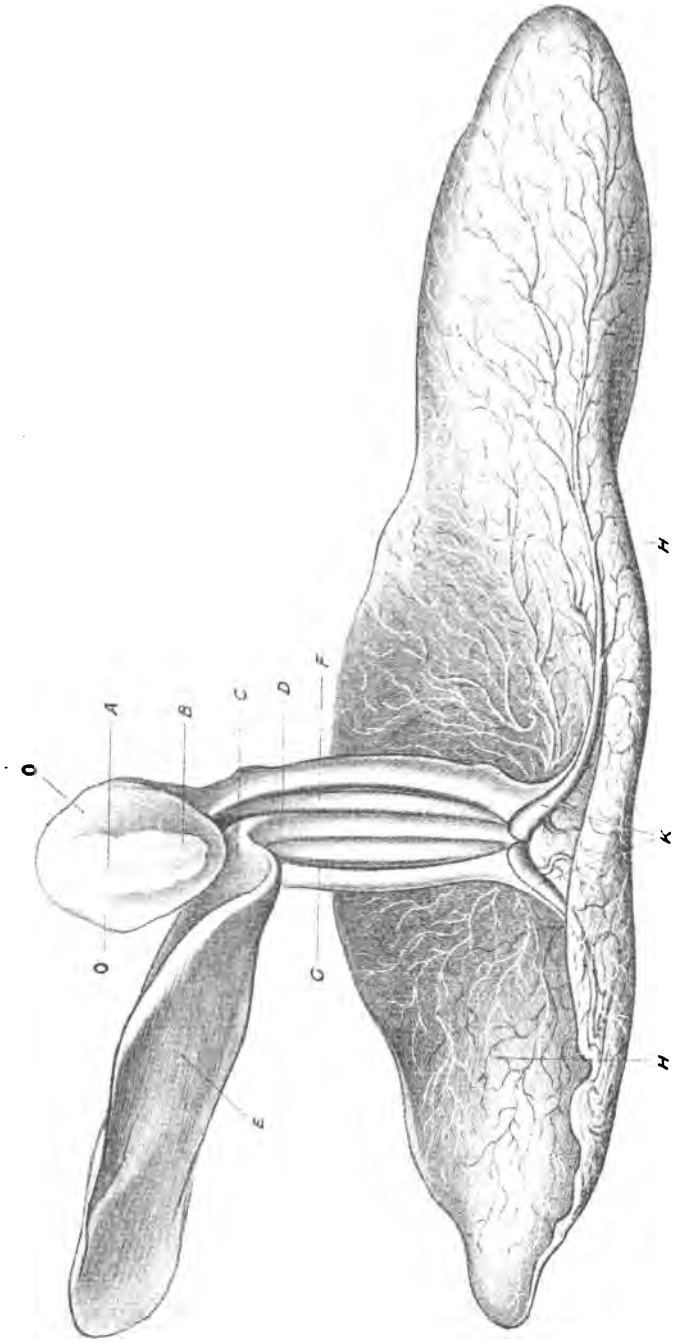


PLATE II.

FIG. 2.

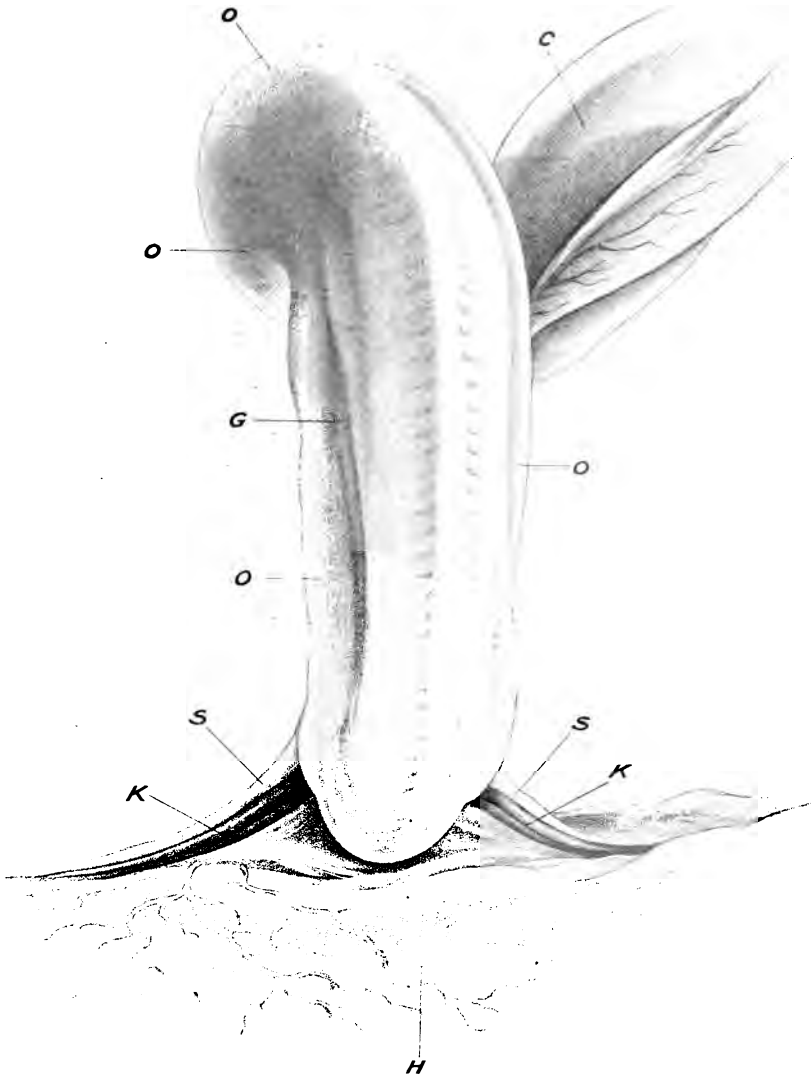


PLATE III.

FIG. 5.

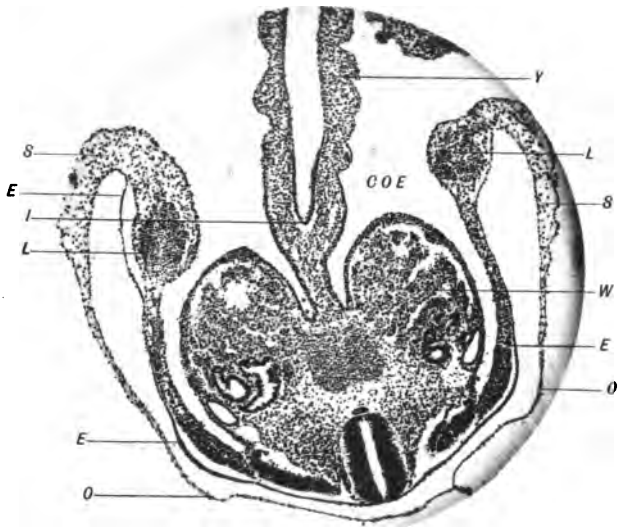


FIG. 6.

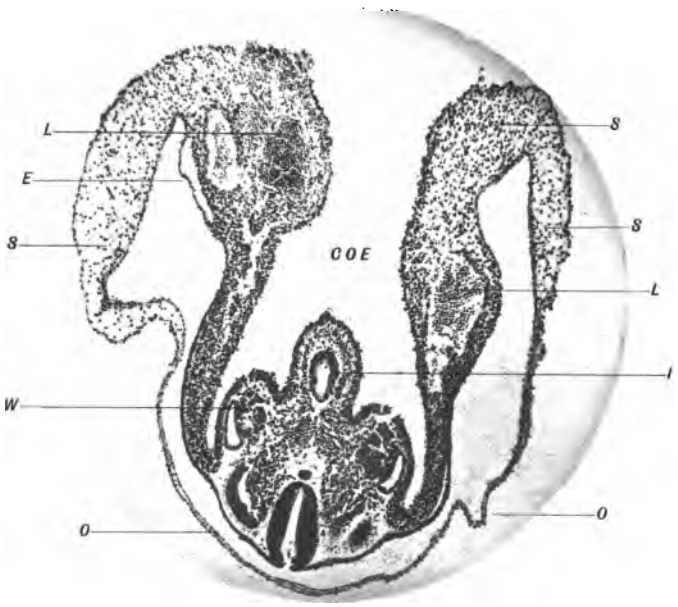


PLATE IV.

FIG. 7.

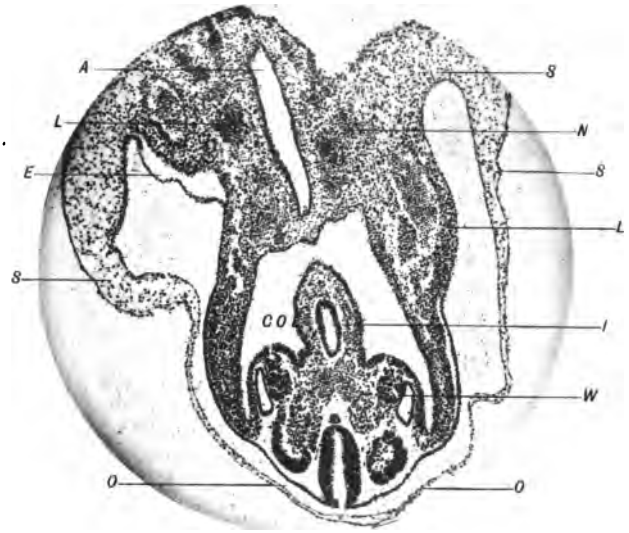


FIG. 8.

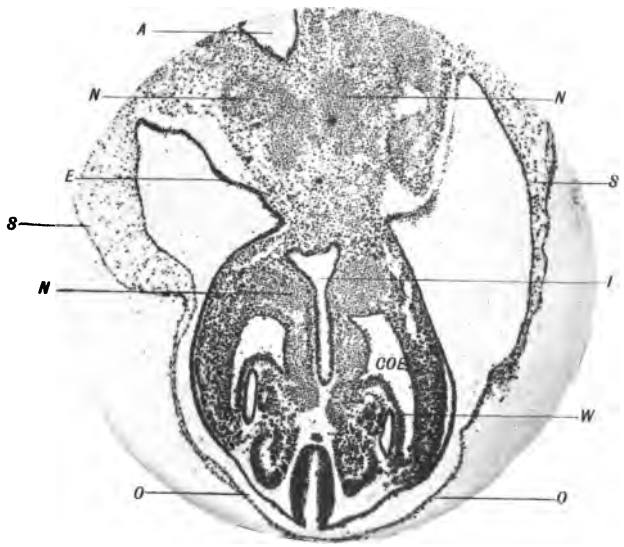


PLATE V.

FIG. 9.

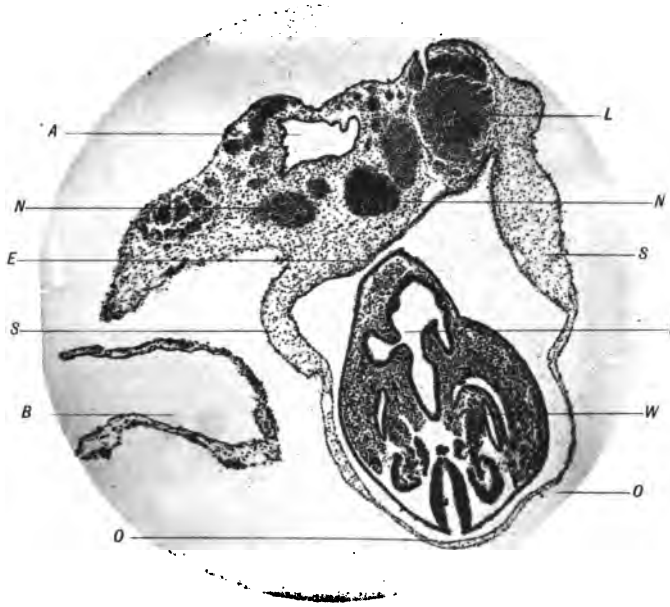


FIG. 10.

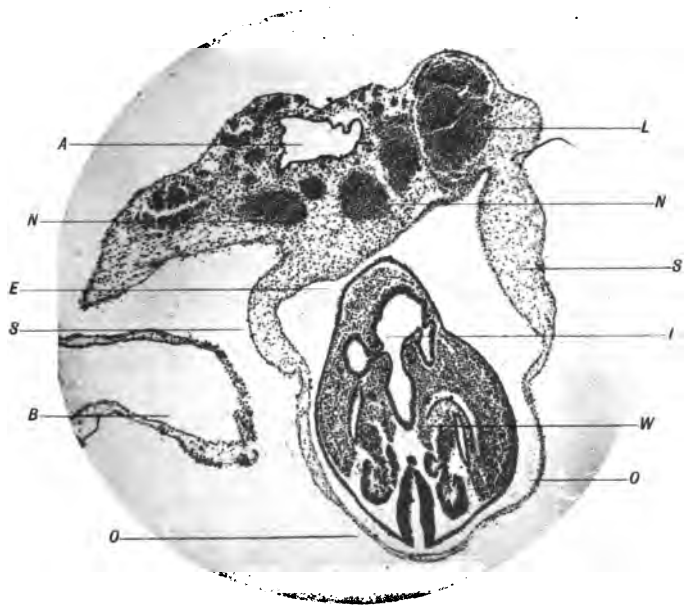


PLATE VI.

FIG. 11.

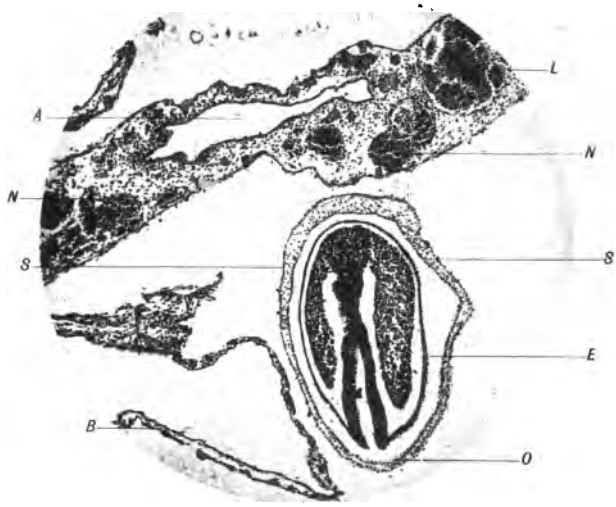


FIG. 12.

PLATE VII.

FIG. 13.

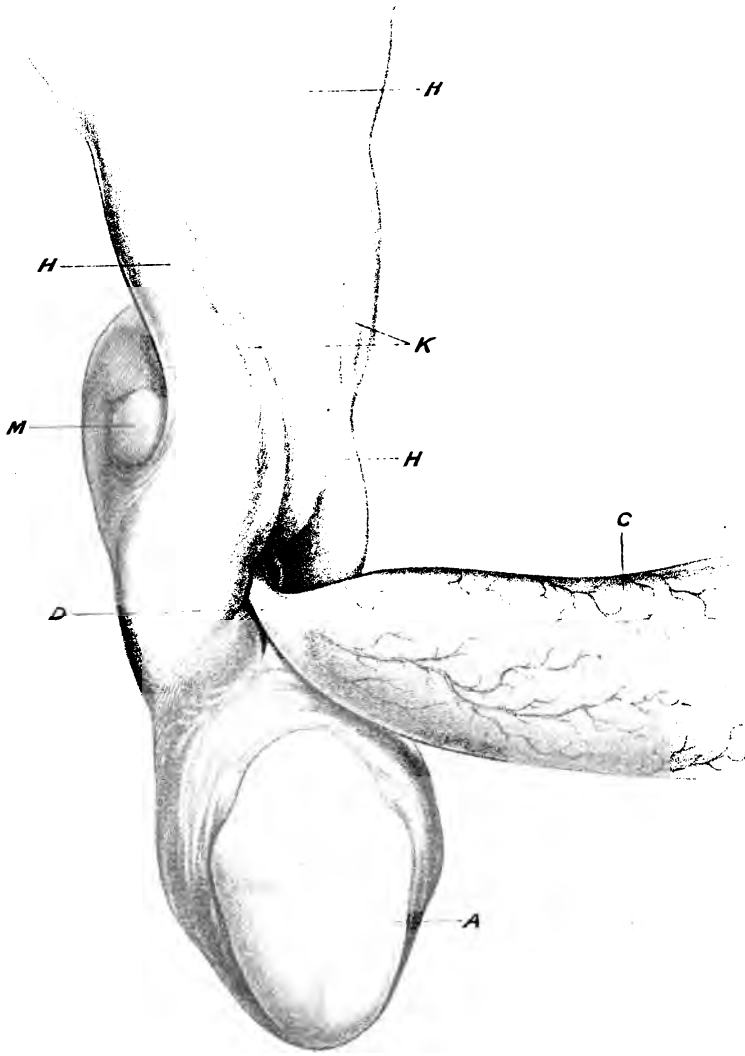


PLATE VIII.

FIG. 14.

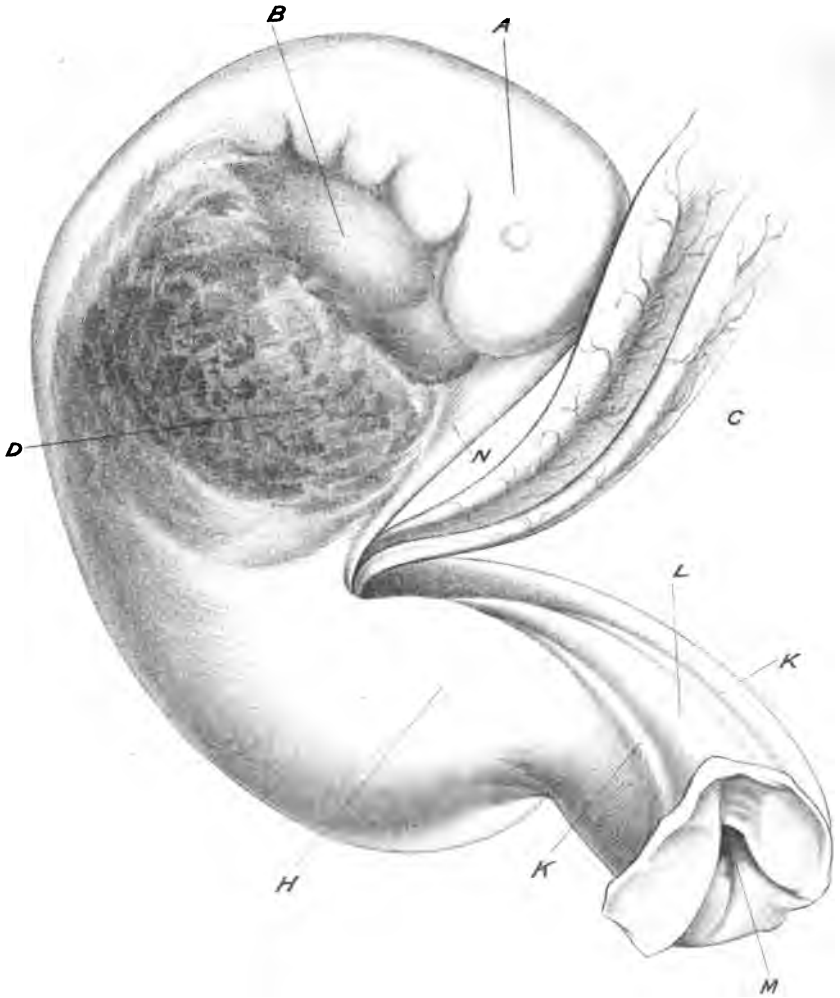


PLATE IX.

FIG. 15.

PLATE X.

FIG. 16.

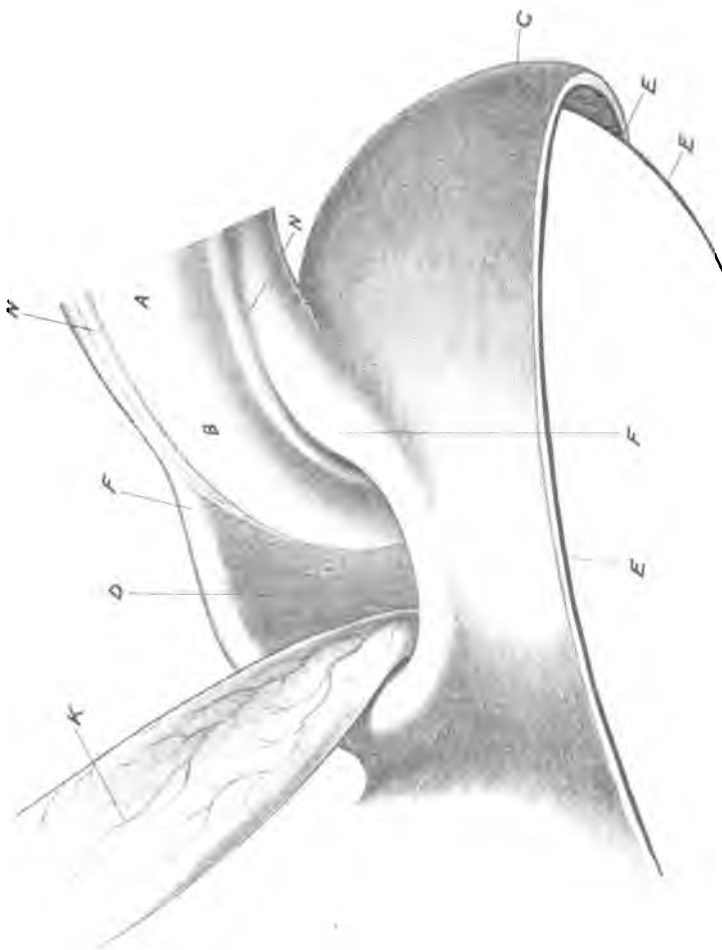


PLATE XI.

FIG. 17.

PLATE XII.

FIG. 19.

FIG. 20.

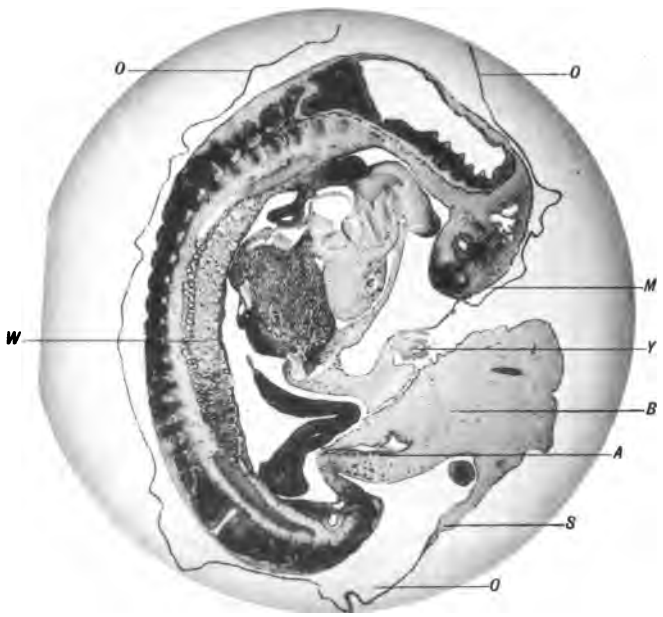


PLATE XIII.

FIG. 21.

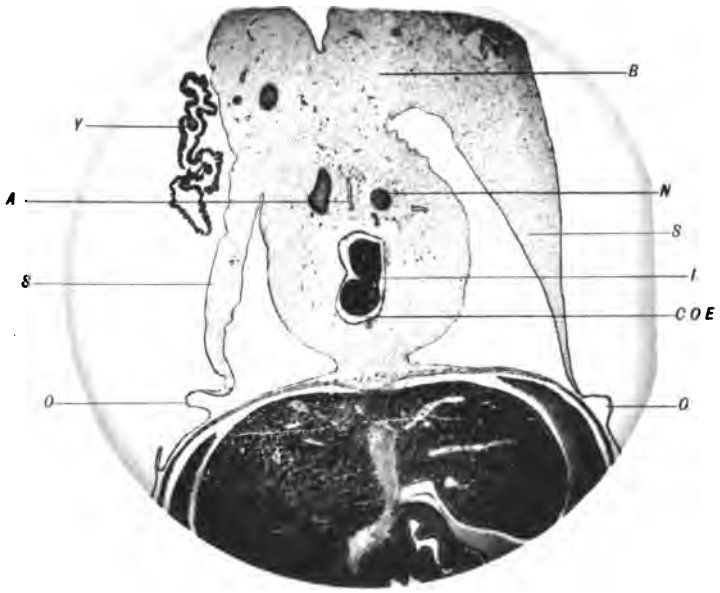
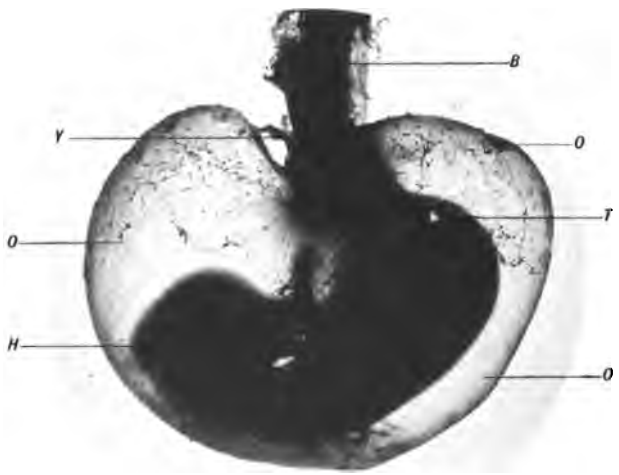


FIG. 22.



brane which here surrounds the cord, but which is altogether separate from it, consists of an epiblastic layer, E, on the inner side and of an outer mesoblastic layer, C, both of which are directly continuous with the same structures in the cord at its distal end. As development proceeds the mesoblastic outer surface of the amnion sac becomes gradually opposed to the inner mesoblastic surface of the chorion, with which it ultimately comes in contact, and to which it is attached by jelly-like connective tissue; but from which it can at all times be easily separated.

Outside the true amnion sac, between it and the false amnion, or chorion, the vascular mesoblastic Bauchstiel, or ventral string, which is continued onwards from the true umbilical cord, spreads out as a vascular mass over the inner surface of the chorion, and the distribution of its blood vessels to the chorionic villi may be partial or general, according to the type of the animal. The outer surface of the true amnion thus covers the placental distribution of these blood vessels, and it is always possible to strip off this amnion membrane from such a placenta; but the amnion cannot be stripped off further than its own attachment to the distal end of the umbilical cord, because, as already described, the amnion membrane is morphologically and structurally a part of the cord itself.

The general conclusion drawn from this investigation is, that the umbilical cord has not at any period of its existence a tubular investment of the amnion.

Meeting IX.—July 4th, 1900

MR A. GORDON MILLER, *President, in the Chair*

I. EXHIBITION OF PATIENTS

1. *Dr Norman Walker* exhibited—

(*a*) A woman with an affection of the skin of the nose closely resembling LUPUS ERYTHEMATOSUS. The lesion was however asymmetrical and the development had been more rapid than usual in that disease. The case was a good illustration of the importance of thorough examination, for on examining the mouth a typical syphilitic ulcer of the palate was discovered.

(*b*) A case of RUPIA, which he agreed with Mr Hutchinson was by no means a common syphilitic lesion. The patient was a child aged 6, and had other marks of congenital syphilis. The lesions themselves were very typical and the nummular scars left, even more so.

(*c*) A case of RINGWORM OF THE HEAD. When first seen the case presented no peculiarities, but under treatment the large nodules disappeared and their place was taken by very numerous small "tubercles"—to use the old denomination. The fungus was of the "rosary" variety.

2. *Mr J. W. Dowden* exhibited a boy after operation for TUBERCULAR DISEASE OF UPPER ULNAR EPIPHYSIS. A boy, aged 5 years, came to the Sick Children's Hospital a little more than a year ago with tubercular disease of the upper end of the ulna. The cavity was opened up and scraped out in August of last year, and as a sinus had not healed by October, again the condition was treated by scraping. On this occasion, however, it was found that the disease invaded the joint, and a portion of articular cartilage of the ulna about the size of a sixpence was removed, and pure carbolic acid applied. The wound healed, but again opened and required a third operation in February; the cavity this time did not communicate with the joint, and the bone around was becoming sclerosed. The condition is now healed, the bone has fallen in, and what is the most gratifying of the results is the unimpaired action in every respect of the elbow joint.

3. *Dr Logan Turner* exhibited a case of ASTHMA ASSOCIATED WITH CHRONIC SUPPURATION IN THE LEFT FRONTAL AND MAXILLARY SINUSES: DISAPPEARANCE OF THE ASTHMA AFTER RADICAL OPERATION UPON THE SINUSES.

Mr H., æt. , was first seen on Nov. 3rd, 1899, having been sent by Dr Brown Darling, who suspected suppuration in one of the nasal accessory sinuses. This suspicion was confirmed, pus being diagnosed in the left antrum and left frontal sinus. Patient had suffered from purulent nasal discharge, chiefly from the left nostril, for more than one year. After an attack of influenza in the autumn, three months before examination, asthma of a somewhat severe nature supervened. A considerable quantity of discharge passed downwards into the throat. On Nov. 17th the left antrum was opened through canine fossa, curetted and plugged. A month later the left frontal sinus was opened, curetted and plugged after a free opening had been made downwards into the nose. The skin incision was accurately sutured, the end of worsted plug being brought out through a small button-hole slit in upper, inner angle of upper eyelid.

The plug was removed on the eighth day. Suppuration ceased entirely after these operations. After the operation upon the frontal sinus with cessation of all purulent discharge from nose, the asthmatic attacks began to abate. Unfortunately a second attack of influenza again caused an exacerbation of it, but these gradually diminished and at this date (July 4th, 1900), the patient remains perfectly well.

4. *Dr W. Russell* exhibited a case of MYXŒDEMA IN THE MALE. As the condition was comparatively rare in men, he thought the members might like to see the patient before treatment commenced.

II. Exhibition of Pathological Specimens

1. *Dr W. Russell* exhibited—

(*a*) A BRAIN FROM A RECENT CASE OF HEMIPLEGIA AND APHASIA which had lasted for about two months. There was a large area of softening in the region of the internal capsule and the parts external to it, caused by thrombosis due to extreme atheroma of cerebral vessels.

(*b*) A BRAIN FROM A LONG-STANDING CASE OF HEMIPLEGIA AND APHASIA, with a very large multiloculated cyst in much the same position as the softened area in the preceding case. It was due to embolism from an acute endocarditis. The patient came home from the East some twelve years ago with the hemiplegia and aphasia, and was thought to have had "sunstroke!"

2. *Dr Alexander Bruce* exhibited a BRAIN FROM A CASE OF APHASIA.

3. *Dr Pearson* exhibited for *Dr G. A. Gibson*—

(*a*) Some slides kindly lent by Dr Patrick Manson, demonstrating the presence of the FILARIA NOCTURNA in the mosquito. One of the slides showed the filariæ in the proboscis of the insect, thereby affording strong evidence of the proof of Dr Manson's mosquito theory of infection in malaria, as well as in filariasis.

(*b*) The THORACIC VISCERA of a patient in whom a persistent arterial duct had been diagnosed during life. *Post-mortem* examination showed that the ductus was patent, and that there was no other cardiac lesion. The case has been fully described in a clinical lecture by Dr Gibson, which appeared

in the *Edinburgh Medical Journal*, new series, 1900, vol. xliii. p. 1.

4. *Dr Harvey Littlejohn* exhibited—

(*a*) Specimen of TRAUMATIC RUPTURE of the heart.

A man, æt. 20, was found dead in the workings of a coal pit. His fellow-workmen asserted that he had died from injuries received from being caught between the buffers of two coal wagons. The Company, owing to the absence of external signs of injury, asserted that death had resulted from natural causes.

At the *post-mortem* no marks of external injury were to be seen. The heart was found ruptured. The rupture being in the right auricle 1½ inches in length.

The ribs were not fractured and there were no other injuries in the body. The heart was normal and healthy.

(*b*) The stomachs from two cases of CARBOLIC ACID POISONING.

In one case, 10 ounces of a strong solution of pure carbolic acid was taken, and the stomach presented the characteristic appearances.

In the other, an indefinite quantity, probably small, of a crude carbolic acid solution, used for cleaning boilers, was taken accidentally in mistake for lager beer. Death occurred in twenty minutes. The stomach was corroded especially at the pyloric end, but had not the characteristic appearance or leathery consistence usually found in carbolic acid poisoning.

(*c*) The liver, uterus and stomach from a case of ACUTE YELLOW ATROPHY.

A woman, æt. 19, unmarried, was pregnant and much depressed in consequence of the father of the child refusing to marry her. About a fortnight before labour commenced she suffered from constant vomiting and inability to take nourishment. She became very markedly jaundiced. She was delivered by means of forceps on account of inertia uteri. Thirty-six hours after the birth of the child, which was alive and healthy, she died suddenly.

At the *post-mortem* numerous small hæmorrhages were seen in the skin and throughout the body and internal organs. The whole body was deeply jaundiced. The liver was atrophied and the liver cells showed advanced fatty degenera-

tion. The kidneys and heart were also undergoing fatty degeneration.

The appearances were consistent with phosphorus poisoning, but Mitscherlich's process, carried out on the contents of the stomach, failed to disclose the presence of phosphorus.

The stomach walls were softened, gelatinous, and near the cardiac end had given way as a result of *post-mortem* digestion.

The uterus presented a normal appearance and showed no trace of any puerperal septic process.

5. *Dr R. A. Fleming* exhibited—

(*a*) TUBERCULAR PERICARDITIS from a patient aged 45, who died of miliary tuberculosis involving lungs, liver, kidneys, spleen, etc. Dr Fleming stated that it was the only specimen he had met with in over a thousand *post-mortem* examinations, and expressed the opinion that it was of very rare occurrence at least in this country.

(*b*) TWO HEARTS WITH PATENT FORAMEN OVALE. The first belonged to the collection of pathological specimens made by the late Dr Rutherford Haldane, and was a very fine example of an unusually large interauricular opening.

The second closely resembled at first sight the condition of things seen in many ordinary adult hearts, only that the valvular flap was open and not closed.

(*c*) FATTY DEGENERATION OF THE HEART almost entirely confined to the left ventricular wall. It belonged to a man aged 56, who died in Dr Affleck's ward of pernicious anæmia, and was an unusually well marked example of the condition.

6. *Mr J. W. Dowden* exhibited a specimen of TORSION OF THE SPERMATIC CORD WITH STRANGULATION OF UNDESCENDED TESTICLE.

A boy, aged 14 months, was brought to the Sick Children's Hospital with the following history. Five days before the child suddenly became sick and vomited, all night he was cross and restless, and the next day it was noticed that sitting up caused him pain. On the two succeeding days he was constipated, but some opening medicine relieved the bowels. The left thigh was noticed to be kept in the flexed position. The day before admission a small lump was noticed in the left groin very tender in character.

On examination the child was obviously ill, the left thigh was flexed, and in the region of the external abdominal ring was a rounded swelling about the size of a small hen's egg, the skin over it was normal in appearance. Temperature was 100·6 F. Palpation revealed a firm tense tumour slightly elastic, but in places a sense of lobulation could be made out. The swelling was very painful. The testicle on that side was absent from the scrotum. With such a history the treatment was obvious—exploratory incision—in case there might be a partial strangulation of gut, though the probability was the condition was testicular, possibly orchitis.

The operation was performed within an hour. After the overlying structures were incised a rounded tense swelling was reached evidently containing fluid; this was opened, about a desert spoonful of blood stained serum escaping. The testicle lay in the sac, of normal size but a dark bluish colour, and above it in lobulations lay the cord, resembling in appearance the collapsed gut, after severe strangulation of intestine. It was found that the testicle could be turned from right to left, so undoing three twists on the cord, a ligature was placed on this structure high up and the cord and testicle removed. Recovery was uneventful.

7. *Dr Alexander James* exhibited a TUMOUR ADHERENT TO THE ABDOMINAL AORTA, which had during life closely simulated an aneurism. The tumour was removed from the body of a man, 35 years of age, who had been a soldier, who had had syphilis, and who had had atheromatous arteries. Double aortic valvular disease, and a very much hypertrophied heart. During life the tumour showed not only pulsation but distinctly expansile pulsation. The two points which during life however had militated against the idea of an aneurism were first that it felt more fixed than an aneurism does, and secondly that there was no change from the normal as regards the pulsation in the femoral arteries.

On *post-mortem* examination a mass of cancer was found growing from the retroperitoneal tissue, encircling the aorta for the greater part of its circumference, and incorporated as it were in its outer coat. The mass was excessively vascular.

III. Original Communicatons

1. CLINICAL NOTE ON RESIDUAL URINE IN CASES OF ENLARGED PROSTATE

By A. G. Miller, M.D., F.R.C.S.Ed., Consulting Surgeon to the Royal Infirmary, and Examiner in Clinical Surgery to the Edinburgh University; President of the Medico-Chirurgical Society

I SUPPOSE that all surgeons will admit that an enlarged prostate of itself causes no harm, and often gives rise to no symptoms. Trouble and danger commence with the residual urine, which, if it increase, gives rise to retention, catheterism, and other serious consequences.

The symptoms that are generally attributed to the enlarged prostate, such as frequency of micturition and so-called incontinence, are really due to the retained urine.

The object of my paper this evening is to draw attention to this dangerous residual urine, to discuss its cause, and to suggest a simple method of treatment, in early cases, that occurred to me in connection with a case which I will relate.

For such cases, hitherto, so far as my information goes, the only treatment available has been the judicious use of the catheter—unless the prostate can be reduced in size, or the bladder stimulated by the administration of ergot—a method of treatment suggested by me some years ago, and which I have found beneficial in cases in which the prostate is soft and where there is hæmorrhage.

A few years ago a very hale and active old doctor consulted me regarding his condition, and propounded the question of the necessity for the catheter in his own case, to prevent increase of the residual urine that he had discovered to exist already to the amount of three ounces. We discussed his case thoroughly with the knowledge, on both sides, of the risks connected with catheterism—however carefully conducted. It occurred to me to say to my friend, "seeing there are these risks, why not first make a special effort to get rid of the residual urine by voluntary evacuation." I suggested that, after he had passed water in his usual manner, he should wait for a short time and then try again, and insist on the evacuation of some more urine. My old friend (he was about seventy) consented to try the plan, and to let me know how he succeeded. He returned in

some weeks saying that he had already benefited considerably. He reported that at first he got rid, at the second effort, of one or two ounces of urine. After a time the quantity was reduced to one ounce, or half an ounce. At the same time he had to get up less frequently at night, and passed his water in a better and more forcible stream at all times. His last report, in a letter which I have, was, that he practised the second effort only night and morning, that the quantity evacuated was quite small—half an ounce or so—and that he was quite contented with his condition.[1]

When I was talking over the doctor's condition with him the view naturally presented itself that the bladder was not emptied thoroughly because sufficient effort was not made to do so, and that a little more force applied was perhaps all that was necessary to completely empty the bladder. If the urine can flow at all it ought to be possible to empty the bladder, and if the act stops short of complete evacuation there must be some reason for this. This suggests the question—Why does an old man with an enlarged prostate not empty his bladder completely? Before answering this, however, let us consider the condition of a man with an enlarged prostate. The man being advanced in years is not so muscular as he was when younger. His muscle tone, and consequently bladder tone, are not so good as they once were. If we take it as granted that there is diminution of muscle power, added to the obstruction from the enlarged prostate, we have two important factors for the production of residual urine. It has occurred to me, however, that there is, or may be, another factor present which has not hitherto been recognised. I have thought of it under various names, such as laziness, indifference, and impatience. Perhaps it varies in different persons. Perhaps it is complex, more or less, in all.

The condition of mind or body to which I thus refer is that which makes the person stop (short of complete emptying of his bladder) whenever he has the sensation of relief, perhaps because he wants to get back to his warm bed as soon as possible, or because he wants to get out of the urinal where he has been standing with damping feet for some time. Perhaps he stops merely because he has got rid of the feeling of distention, even

[1] He assisted the second effort sometimes by standing at a fixed basin in his room and turning on the water-tap.

though he may know that he will be all the sooner obliged to pass water again.

My object is, as you will see, to point out that impatience or hurry, indifference to consequences (unknown doubtless to most), and something like disinclination to make an effort, are the causes of the failure to empty the bladder, and therefore of the presence of the residual urine. To put the matter into a single sentence—*The cause of residual urine is neglect to empty the bladder.* I admit that not infrequently the word "inability" might be substituted for "neglect" with accuracy. But I submit at the same time that even in these cases neglect has probably begun what inability keeps going, and increases.[1]

In order that I may still further enforce this view, let me refer briefly to the way in which the bladder is emptied naturally and physiologically. There is a twofold action—first, the relaxation of the muscle or muscles which close the cavity; and, second, the contraction of the bladder itself, aided by atmospheric pressure, and sometimes by the abdominal and pelvic muscles. These muscular actions are partly voluntary and partly involuntary.

Now I submit that the whole process, though complex, partakes more of a *relaxation* than of an *effort*; of course I mean under ordinary circumstances. Is it not so? We experience a sensation of relief after passing water, and the relief is all the more pleasant that it is without exertion. In other words, we generally pass urine, after the flow is started, by the contraction of the bladder only, aided by atmospheric pressure. Apply this now to the case of the man with an enlarged prostate. He has been accustomed for sixty years or more to pass his water at will without any trouble. He has hitherto experienced relief and comfort without any exertion. Why should he exert himself now? Well, he doesn't. What is the consequence? Just what we have seen. He stops short of completely emptying his bladder, and residual urine is the result. It comes to this —it is very much a matter of habit. The man with residual urine has lapsed into a habit of not emptying his bladder. No

[1] It must be remembered that, as I said formerly, I am not referring to old-standing and advanced cases, but to those in which the formation of residual urine is *commencing*.

doubt because emptying it has become more difficult. Under these circumstances I hold that the rational treatment is to break off the habit while this is possible, namely, at the beginning, and to establish instead a practice of emptying the bladder regularly.

I may be told that prostatic patients do exert themselves, and have to strain, sometimes very severely, when passing water. That is so. But these are advanced cases. The straining comes too late. The bladder has lost its power and function, and the work has to be done largely by the abdominal muscles. What I suggest ought to prevent this condition of matters by training the bladder to empty itself, and so to keep it healthy, in good tone, and functionally active.

Sir Henry Thompson,[1] in his lectures on the surgery of the urinary organs, when speaking of atony of the bladder, says that this is most frequently caused by enlargement of the prostate, and in speaking of residual urine he says that a habit of not quite emptying the bladder may be acquired. In these statements he seems to support my view of the cause of residual urine in cases of enlarged prostate. When, however, he deals with the subject of enlarged prostate,[2] he states that hypertrophy with dilatation is the most common condition. In this latter opinion Mr Reginald Harrison coincides,[3] and he is of opinion that the residual urine is most commonly due to sacculation of the bladder, the mucous membrane being herniated between thickened bands of the muscular layer of the bladder.

This apparent contradiction is, I think, to be explained by the supposition that these latter statements refer to advanced cases of prostatic disease. On the other hand, my present argument refers to early cases—those in which residual urine is only commencing to form.

I would put my deductions as two :—

1. Residual urine is due at first to neglect to empty the bladder.

2. Accumulation is due to this becoming a habit.

The practical application is that men advanced in years who are beginning to have prostatic symptoms should make a point of emptying their bladder thoroughly.

[1] "Surgery of the Urinary Organs," 1884, p. 99.
[2] "Diseases of the Prostate," 1886, p. 108.
[3] *Lancet*, 5th May 1900.

My advice is: after passing water in the usual way, wait a minute or two and then try again. Practise this as frequently as possible, till only a very small quantity can be squeezed out (ʒ i). When that point has been reached, twice or thrice daily may suffice. How does this act? In two ways. First, the small residuum of urine that may result from a careless act of micturition is got rid of. In the second place, the bladder tone is improved, and its function restored. One might add that the habit of completely emptying the bladder is acquired. A still more important result is that the residual urine being diminished cannot possibly increase. *Obsta principiis* is a good working principle.

I do not bring this forward as a cure, or as a means of preventing residual urine from forming in every case of enlarged prostate. I merely suggest a safe and simple procedure, which has apparently checked the increase of residual urine in several cases, and which may in some others at least postpone the almost inevitable catheter.

2. CASE OF ACUTE ASCENDING PARALYSIS DUE TO INFLUENZA

By ALEXANDER JAMES, M.D., F.R.C.P.Ed., Physician to the Royal Infirmary, Edinburgh

POST-MORTEM EXAMINATION

By ROBT. A. FLEMING, M.D., F.R.C.P.Ed., Pathologist to the Royal Infirmary, Edinburgh

THE influenza organism is one which can affect injuriously practically all the systems of the human body. When it affects the nervous system it can bring about a manifestation of one or other of practically all the symptoms of nervous system diseases. It can produce mental disturbances—paraplegias — hemiplegias — monoplegias. It can pick out certain strands in the cerebro-spinal axis and can induce the symptoms of a lateral sclerosis — a progressive muscular atrophy or even a locomotor ataxy. It can produce a polyneuritis, and it can pick out certain nerves for the manifestation of its injurious effects. Oculo-motor paralysis we used to be told—wherever we find it—we must ascribe to syphilis. I believe we must modify our opinion since we have made the acquaintance of influenza.

During the winter and spring of this year I had the opportunity of witnessing the effects of the actions of the influenza poison on the nervous system in a manner which I had read of, but never had seen before. This was as inducing a rapidly progressive paralysis of limbs and trunk from below upwards in the manner described as Landry's paralysis. I saw this in three cases, and curiously they all occurred within a few weeks of one another. I shall only give the details of one—the one in which the diagnosis was verified by *post-mortem* examination, but I should like to say that this one as regards age and sex of patient, symptoms and course, was almost identical with one of the other two.

Mary M., aged twenty-three, a housekeeper, residing in London, but on a visit to friends in Edinburgh, was admitted to Ward 33, January 31st, 1900. Her complaints were pains specially in the head and back, and she looked very ill and was at times delirious. Her illness had begun two weeks previously.

Her family history was not satisfactory; Bright's disease and heart disease were present, and there was also a family history of insanity. Her home was comfortable, her work not too hard, and she had been careful and regular as regards diet.

Previous illnesses.—She had had measles when a child, and in May last she seems to have had an attack of influenza, the symptoms being mainly headache and pain in the back. She recovered readily, and three months ago, a small fatty tumour was removed from her back. From this she recovered readily.

Present illness.—This began a fortnight ago, just as the wound in the back was healed. She felt she had another attack of influenza and complained of headache, and had vomiting and constipation. A week before admission the headaches became very severe, and she became very weak and very sleepless at night, calling out continuously and apparently meaninglessly, so as to require constant attendance. As day after day passed, she remained much in the same condition, complaining of headache and pains all over and being so restless and noisy at night that it was thought by her friends that she would have to be sent to an asylum. Now and again for short intervals she became quieter and fairly sensible, and then she often complained of a lump rising in her throat.

On the morning of January 31st I saw her for the first time. Temp. 103°; pulse 130, regular and of fair strength; resp. 22. She would not answer questions, and kept crying out mostly "Oh, dear!" and "Oh!" She lay on her back, and did not move her legs or arms, and I ascertained that the fact that she was not moving her legs at all had been noted the day before. The urine and fæces were being now passed in bed, but to some extent she was yet indicating when she required the bed-pan by calling out and by restless movements of the head.

Cutaneous sensibility to touch and pain seemed all over little impaired. Her pupils were distinctly dilated. Voluntary motor power in the legs and arms appeared lost, and the muscles were flaccid.

The abdominal and lower costal respiratory movements were not well marked, but there occurred no distinct diaphragmatic paralysis.

By questioning the friends and by watching the patient I tried to ascertain the presence of Cheyne Stokes breathing. It was certainly present, but curiously it seemed to be absent when she was deeply asleep.

Superficial and deep reflexes in legs and arms were absent.

Swallowing seemed to be becoming impaired to-day.

Realising that this was a very anxious case, I advised her admission to the Infirmary at once.

State on admission.—She appeared more sensible, and complained of the lump in the throat, and then suddenly she began to breathe very rapidly—about 60 per minute for 30 seconds—and then became quieter and spoke of pain behind the sternum.

Her speech was somewhat slurring—the upper lip not moving well.

Swallowing was performed with some difficulty, tending to cause choking; and paralysis of arms and legs was noticed.

At 8 P.M. she appeared rather better, but said she could not move her legs or arms, and her muscles were flaccid. Temp. 99·4; pulse 130. Shortly after this curious nodding and shaking movements of the head occurred, and she began to cry out "murder."

Digitalis and chloral were given, and soon she fell asleep,

and awoke about 11 P.M., expressing herself as better, and looking more sensible.

She went to sleep again, and about 1 A.M., the nurse noticed that her breathing was very quiet. In a few minutes she quietly died.

Post-mortem Examination and Microscopic Examination

The *post-mortem* examination was performed the day after death. The body was fairly well nourished, and the muscularity fair. Lividity was slight and rigidity marked.

Thorax.—Heart weighed 8 oz. The left ventricle was firmly contracted, and the right was flaccid. Arterial valves were competent. Orifices were fairly healthy. Muscle of heart was fairly healthy in appearance, although somewhat flabby.

Lungs.—Right, 15 oz.; *Left*, 12 oz. Lower lobes were slightly œdematous and congested, and there was commencing broncho-pneumonia near the root of the left lung.

Abdomen.—Liver, 3 lbs. 2 oz., showed marked cloudy swelling; and *Kidneys—Left*, 5 oz.; *Right*, 6 oz.—showed similar changes.

Spleen, 3½ oz. Although it was of small size it showed distinct acute congestion with numerous small hæmorrhages scattered over the cut surfaces.

The other abdominal organs were healthy.

Nervous system.—Brain, 3 lbs. 2 oz. Dura mater was firmly adherent posteriorly over vertex. Sinuses contained no clot. The convolutions of both hemispheres were slightly flattened, most markedly perhaps over the right Rolandic areas.

Pia-arachnoid was somewhat thickened. On section the brain was found to be œdematous, and the grey matter of cerebrum, pons, medulla, and cord was markedly congested. There were no visible hæmorrhages, and further than the congestion of grey matter the spinal cord was healthy.

Microscopic appearances.—1. There was no degeneration in any of the tracts of the spinal cord, although Lissauer's tract appeared to stain somewhat faintly in many of the segments of the cord examined, including the lumbar and cervical enlargements. Weigert's chrome-alum copper solution and osmic acid were used for fixing and staining the cord, with the usual counterstains.

2. There was great engorgement of the vessels in the grey matter of the cord, and especially the anterior cornua; and portions of the cord fixed in corrosive sublimate and embedded in paraffin showed numerous minute hæmorrhages, which perhaps should be more correctly described as microscopic diapedeses of red blood corpuscles. Many leucocytes or lymphoid cells were also noted in the grey matter of the cord, and were certainly in abnormal numbers around the multipolar cells, especially of the lumbar and cervical enlargements. The minute hæmorrhages were also more numerous in the enlargements.

3. The multipolar cells of the anterior cornua were shrunken, were somewhat deeply stained with Toluidin blue and other basic dyes, and the nuclei were displaced towards the periphery of many of the cells. A small proportion of the multipolar cells were normal, and these were most numerous in the upper cervical segments. The cells of Clarke's vesicular columns were unaffected.

4. Many of the cells of the nuclei in the medulla were swollen, stained feebly and homogeneously with Toluidin blue, showing marked chromatolysis and disappearance of much of the chromatic substance altogether from the cells. An attempt was made to locate the nuclei which showed these cells in greatest number; but all that can be stated is, that the change was not limited to motor nuclei alone, and that the nuclei in the upper third of the medulla were less affected than those in the lower two-thirds. The engorgement of vessels and minute microscopic hæmorrhages were present in the medulla, although the hæmorrhages were not so numerous as in the cord.

A weaker solution of corrosive sublimate than intended was used for fixing the portions of cord and medulla, but the fact that certain cells, such as those in Clarke's vesicular column, were neither shrunken nor swollen but fixed and stained normally, obviates to some degree the criticism that the cell changes were artificially produced.

Most unfortunately, the nerves were not obtained for examination, but the nerve roots appeared to be healthy.

Hæmorrhages, and swelling, and atrophy of nerve-cells, have been recorded in several cases by Gombault, Leyden, and

others, and in a number of equally carefully investigated cases no change of any kind has been recognised.

Modern methods are unquestionably throwing more light on the intricate problem of cell-changes, and it seems hardly credible that a toxine capable of completely and incurably paralysing nerve-cells can fail to produce a recognisable interference with cell structure.

3. A CASE OF PARALYSIS OF THE VERTICAL MOVEMENTS OF THE EYEBALLS WITH CONSERVATION OF THE LATERAL MOVEMENTS

By ALEXANDER BRUCE, M.D., F.R.C.P.Ed., Assistant-Physician to the Royal Infirmary and Lecturer on Practice of Medicine at Surgeons' Hall

PARALYSIS of the conjugate deviation of both eyes to one side is a condition of comparatively frequent occurrence and is easy of explanation on anatomical grounds, for it is now well established that the sixth nucleus in the pons is not merely the nucleus of the sixth nerve of its own side, but also, through the third nerve of the opposite side, supplies the internal rectus. The sixth nucleus thus innervates the external rectus of its own and the internal rectus of the opposite side. Its action is therefore to draw both eyes to its own side, much in the same way as the hand of a coachman driving a pair of horses in pulling one rein draws both horses to the same side. Paralysis of one sixth nucleus simply paralyses this power of moving both eyes to the same side, while leaving the opposite nucleus and the nucleus for convergence free to act. There is also a cortical centre for conjugate lateral deviation which may be stimulated or paralysed (if only for a time) in cortical lesions. Paralysis of both sixth nuclei will produce complete inability to move the eyes to either side, but will leave the vertical movements and the power of accommodation quite intact. This condition of bilateral paralysis is certainly not common, but its mechanism is well understood.

On the other hand, the converse condition of loss of the vertical movements with conservation of the lateral movements has been recorded only once or twice. Gowers (Trans. Ophth. Soc., i. 81, p. 117) has described one case in which there

was paralysis of the upward movements of the eyes without affection of the elevators of the eyelids. In this case "a very small tumour was found in the middle line behind the posterior quadrigeminal bodies, damaging these slightly, the velum, and the adjacent part of the inferior vermiform process of the cerebellum" (Gowers, "Diseases of the Nervous System," 2nd edition, vol. ii. p. 186). Wernicke (Berlin, "Klin. Wochenschrift," 1878, p. 154) has described a case of paralysis of the upward and downward movements of both eyes, accompanied by ptosis. In this case the lesion was situated in the optic thalamus and corpus striatum. A similar case, not accessible to me, has been described by Lang and Fitzgerald (Trans. Ophth. Soc., vol. ii. p. 230). So far as I am aware no cortical cerebral lesion has been described producing isolated bilateral paralysis of the vertical movements, nor has any cerebellar lesion been found which confirms the results of physiological experiment. It is well known that experimental lesions of the middle lobe of the cerebellum produce paralysis of the vertical movements of the eyeballs. Ferrier has shown, for example, that electrical stimulation of the upper part of the middle lobe of the cerebellum causes both eyes to move upwards, and that stimulation of the lower part of the same lobe moves the eyes downwards. Destruction of the posterior part of the median lobe produced a tendency to fall backwards. The relations of the middle lobe of the cerebellum to the movements of the eyeballs and to equilibration being established so definitely by experiment, the following case in which the lesion was ascertained *post-mortem* to be *not* in the cerebellum is of interest, as well as from the fact that it appears to establish the influence of another co-ordinating centre for equilibration.

M. H., age 46, single, was admitted on the recommendation of Dr Chalmers of Newhouse to Ward 25, Royal Infirmary, on March 31, 1900. Her history is briefly as follows. In August 1899 she felt "out of sorts" and weak in the legs. After two or three days' rest, however, she continued at her work as a weaver till January 1900, when, after exposure to a severe snow-storm, she had a rigor, an attack of pain, deafness, and tinnitus in the left ear. Her eyesight became weak and dim, so that she could not see to work. The deep-seated pain in the ear and the deafness have continued, much the same as at first. There is no history of vomiting, but there have been occasional

headaches, not of a severe type. On further inquiry it was ascertained that she had fallen from a chair seven years ago and struck her head violently against the wall of the room. She is a total abstainer from alcohol, and has not had syphilis.

There are no sensory abnormalities. Sensibility to touch, temperature and pain are normal. Perception of difference of weights is good. Co-ordination of movements of upper extremities is quite normal. She can touch the tip of the nose with the forefinger when the eyes are shut. She can also bring the forefingers together when the eyes are shut, and can button her clothes with perfect ease. When she stands with the eyes shut, she tends to fall backwards unless supported. In a few days it was found that even when her eyes were open she could not stand erect. There was a constant tendency to fall backwards, or rather to bear backwards even against a supporting hand. When she was placed on a chair and made to lean as far forwards as possible, she could remain in this position only so long as she was allowed to place her hands on her knees. So soon as these were removed, even although she was so far bent forward as would, under ordinary circumstances, have caused her to fall forwards, she immediately straightened herself up and then fell backwards. There never was any tendency to fall either forwards or to one or other side. The same tendency to fall directly backwards appeared when, after being raised in bed, the support was removed. In walking, which was impossible without support, the head and chest were held well back, the heels were brought down to the ground in a slow deliberate manner, the rest of the foot is then brought down with a slow dorsiflexion of the toes. The left leg seems weaker than the right. There were exaggerated knee-jerk, ankle clonus, and patellar clonus on both sides, most marked on the right. Both plantar reflexes were exaggerated, especially on the right side. There was slight difficulty in swallowing and the urine, shortly after admission, was passed involuntarily and unconsciously. Her eyes were examined by Dr Lyon, who reported that the palpebral fissures were fairly widely open, and equal in size. Act of winking performed quite well. *No ptosis. No upward movement of the eyeballs or of the upper eyelids when the patient attempted to look upwards. Downward movement imperfect. Lateral conjugate deviation to both sides perfect. Very slight power of convergence.* (This was

present, and not abnormal, on admission). No nystagmus. On ophthalmoscopic examination the veins of the fundus oculi were large; slight obscuration of the margins of the discs. Vessels nowhere obscured, left disc *more* hyperæmic. Taste and smell were normal. Hearing worse in right ear than in left.

Her further progress was one of gradually increasing hebetude, with occasional cerebral vomiting, especially when raised in bed, involuntary micturition, occasional delusions as to the presence of her mother in the ward. There was a slight tendency to turn the head to the left, and a slight drag of the mouth to the left side. The pupils became dilated, and reacted to light only slightly. There was no ptosis until the day of her death. This occurred on the 24th of April, being preceded by complete coma, a rise to 102° of the temperature, which had previously been normal or subnormal, of the pulse from an average of 75 to a rate of 170, and of respirations from 24 to 40 per minute.

A *post-mortem* examination was secured with difficulty through the exertions of Dr Chalmers, and revealed an interesting and somewhat unexpected state of matters—viz., a tumour which proved on microscopic examination to be a very vascular glioma of the grey matter round the aqueduct of Sylvius, extending from a point midway between the two corpora quadrigemina as high as the third ventricle, into the posterior part of which it projected like half a bean. At the level of the upper part of the third nucleus the tumour had infiltrated practically the whole of the Sylvian grey matter, and had surrounded the cells of the oculo motor nucleus. The lower part of the nucleus, however, was not so completely involved by the infiltration, and the fourth nucleus had escaped entirely. The pons, sixth nucleus, and cerebellum, were quite normal. A second pea-sized glioma was found on the right side of the septum lucidum projecting into the lateral ventricle. Both lateral ventricles were somewhat distended with a clear fluid.

In considering the case at the bedside I was inclined to place the probable site of the lesion in the upper part of the middle lobe of the cerebellum. Taken in conjunction with the case of Gowers, and with the results of experiment, a lesion in this situation seemed more likely to produce the eye symptoms and the peculiar disturbance of equilibration than would a

lesion in the optic thalamus as described by Wernicke. The gradually increasing blindness and hebetude, further interference with the ocular movements, and the terminal coma were attributed to a probable blocking of the aqueduct of sylvius, and a consequent hindrance to the escape of cerebrospinal fluid from the ventricles of the cerebrum. The former part of the diagnosis was wrong, the latter probably right. The symptoms, in so far at least as the eyes were concerned, were probably due to the gradual downward infiltration of the gliomatous tissue, and their mode of onset seems to indicate that the elevators of the globe occupy a higher position than the depressors. With reference to the tendency to retropulsion, I would draw attention to the fact that Hamilton of Aberdeen and Bechterew of St Petersburg have expressed the opinion that the walls of the third ventricle have some influence on equilibration. The free portion of the tumour which projected into the ventricle was in a position to exert pressure on its walls.

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